VOL. 15 JANUARY, 1941 No. 1

THE PSYCHIATRIC QUARTERLY

PUBLISHED BY AUTHORITY OF THE
NEW YORK STATE DEPARTMENT OF MENTAL HYGIENE

The Psychiatric Quarterly, formerly the State Hospital Quarterly, is the official organ of the New York State Department of Mental Hygiene.

Volumes begin with the January number. Annual subscription rate, $2.00 in U. S. and its possessions; $2.50 elsewhere.

Editorial communications, books for review and exchanges should be addressed to the editor, Dr. Richard H. Hutchings, Utica State Hospital, Utica, N. Y.

Business communications, remittances and subscriptions should be addressed to the State Hospitals Press, Utica, N. Y.

Entered as second-class matter April 17, 1917, at the postoffice at Utica, N. Y., under the Act of March 3, 1897.

ALZHEIMER'S DISEASE*

(*An Attempt at Establishing the Adult Type of the Disease*)

BY ARMANDO FERRARO, M. D., AND GEORGE A. JERVIS, M. D.

There is general agreement that Alzheimer's disease is a well-defined clinicopathologic entity. Clinically, it is characterized, first, by a progressive and severe mental deterioration, organic in type, occurring in the presenile age; second, by focal cortical signs such as aphasia, paraphasia, apraxia, etc.; and third, by various neurological signs including hyperactivity of the reflexes, pyramidal tract signs, alteration in the pupillary activity, incoordination, tremors, rigidities. Generalized epileptic seizures are observed as a rule. The underlying brain pathology consists of a severe atrophic process involving mainly the neuron cells, which exhibit the whole range of degenerative changes characteristic of senility. In addition, a very large number of senile plaques and of Alzheimer's neurofibrillary changes is found, while arteriosclerotic alterations are usually absent, or unimportant.

Although numerous contributions have done much to further our knowledge of the clinical and pathologic aspects of Alzheimer's disease, little progress has been made in the study of its etiologic factors. Since the disease was first described in 1906 there has been general agreement that the condition is closely related to the process of senescence. The age of onset in the so-called presenium and the close similarity of the pathologic process to that of senile dementia are good evidence in favor of this opinion. However, many an investigator has recently expressed the stimulating view that a variety of factors, independent of senile conditions, may play a rôle in the causation of the disease. This contention put forward by American authors,[1] has been supported in Germany by Braunmühl,[2] who recently stated that Alzheimer's disease is no early form of senile dementia but rather a condition *sui generis*.

There can be little doubt that the observation of cases of Alzheimer's disease occurring as early as the fourth decade of life is of considerable interest for the problem of the nature of the pathologic condition. It is the purpose of this presentation to put on

*Read at the meeting of the Medical Society of St. Elizabeth's Hospital, Washington, D. C., on April 20, 1940.

record two cases of Alzheimer's disease with onset in the fourth decade (at 33 and 38 years of age, respectively), hence much before the presenile period. The study of this, and a few other cases reported in the literature, indicates that Alzheimer's disease may occur in the adult life. For this variety the term "Adult Type" is suggested.

Case Reports

Case 1. The patient, C. M. C. (No. 309840), a white female, age 38, was admitted to Marcy State Hospital on April 9, 1938.

Little was known of the patient's parents, but it could be established that they had shown no signs of mental or nervous disease. There were two siblings; a sister, aged 34, who had had a psychosis characterized by "depression," from which she had apparently recovered, and a brother who had been a patient in a State hospital for over 10 years, suffering from a mental condition diagnosed dementia præcox. Unfortunately very little information could be obtained concerning the clinical details of these psychoses.

The early history of the patient showed nothing of significance. She had had a common school and business education and worked as a stenographer before her marriage. Her personality showed no abnormal trends. She had never had any serious illness except pneumonia several years before admission. The onset of the psychosis was gradual. The first symptoms were observed in 1933 when the patient was 33. At this time, she showed a definite loss of interest in social activities, was forgetful and inefficient to the point of being unable to take responsibilities in the management of the home. She often also complained of headaches and dizziness. The condition became slowly and progressively worse, and, on August 10, 1937, she was admitted to the Syracuse Psychopathic Hospital. There, she appeared to be intellectually deteriorated. Her answers to questions were inadequate and slow. She was easily confused, and her comprehension was poor. In the attempt to cover gross memory defects, she showed some fabrication. Emotionally, she was euphoric. She had no insight into her condition. At neurological examination, there was slurring of speech and inability to pronounce test phrases. All deep reflexes were overactive, and a questionable Babinski sign was elicited bilaterally. The finger to nose test showed definite incoordination.

The gait was shuffling. The pupils were anisocoric. Laboratory examinations were negative.

On November 4, 1937, she was discharged from the Syracuse Psychopathic Hospital. On her return home, she was unable to do any work and appeared to be in a daze most of the time. When spoken to, she looked blankly and repeated what was said to her.

On April 9, 1938, she was admitted to Marcy State Hospital. Here she was dull, inactive, underproductive and irrelevant. There was stammering of speech and evidence of sensory aphasia. Her emotional reaction was silly and inappropriate. She denied hallucinations. She was completely disoriented. Remote memory was markedly defective, and recent memory and retention were absent. She was unable to read or write. At physical examination, she was well nourished and well developed. The right pupil was larger than the left but both pupils reacted to light and accommodation. All deep reflexes were overactive. Blood pressure was 110/76. Routine laboratory examinations of urine, blood and spinal fluid were negative.

X-ray examination of the skull showed no evidence of pathology. The air encephalogram revealed general dilatation of the ventricles and marked widening of the cerebral sulci diffusely distributed over the entire brain surface.

Following admission, the demential state became progressively worse. During the first months of 1938, she was entirely out of contact with her surroundings. She was unable to comprehend when spoken to. The speech defect became worse; at times she repeated over and over again the same words. She had spells of compulsive laughter. Numerous involuntary movements of the body, choreiform in character, were first observed during this period. In June, 1938, she had a series of severe convulsive seizures following which she became physically weak and bedridden.

The last year of life, the patient failed physically, becoming emaciated. Convulsive seizures continued, increasing in severity and duration. She died on October 12, 1939.

The autopsy was performed five hours after death at the Marcy State Hospital, October 12, 1939, by Dr. G. C. Bower. The brain was fixed in formalin and preserved intact for further study. No significant abnormal finding was reported at post-mortem examina-

tion beyond pulmonary abscesses involving both lobes of the left lung.

The brain weighed 1004 gm. On external examination, there was a marked atrophy of the convolutions with gapping of the sulci. The atrophy was diffusely distributed and symmetrical, being, however, somewhat more marked in the frontal and temporal lobes. There was a slight thickening of the leptomeninges. The basal vessels were normal in size and distribution and showed no atheromatous changes.

On frontal serial sections, the marked atrophy of the convolution was confirmed; the white matter of the cerebral hemispheres was also reduced in size, firm in consistency and without any foci of softening. The basal ganglia were atrophic and both lateral ventricles were considerably dilated. Cerebellum, pons and medulla appeared normal to naked eye examination.

For histologic examination, the following methods were used: Nissl's stain for neuron cells in material fixed in alcohol and embedded in celloidin; Bielschowsky and Braunmühl stains for neurofibrils and senile plaques on frozen sections fixed in formalin; Herxheimer stain for fatty substance; Spielmeyer's stain for myelin sheaths; Holzer, Cajal and Hortega's stains for the various types of glia.

Examination of numerous cortical fields with Nissl's method revealed widespread changes of neuron cells and neuroglia. The gray matter of the cortex appeared everywhere narrower than normal, the boundary between gray and white matter was sharply defined due to the fact that the nuclei of the white matter were apparently increased in number. In the frontal lobe, the normal cortical cyto-architectonics were considerably disturbed, numerous neurons having disappeared. In the frontal polar region, loss of nerve cells was evenly distributed throughout the six layers (Fig. 1) while in the granular cortex the third and fifth layer appeared more severely involved than the other four. However, there was considerable variation in closely adjacent microscopic fields even within the same cyto-architectural area. Thus, in the frontogranular region, islands of remarkably well-preserved pyramidal cells of the third and fifth layer could be observed in areas in which these two layers were severely affected. A diffuse proliferation of

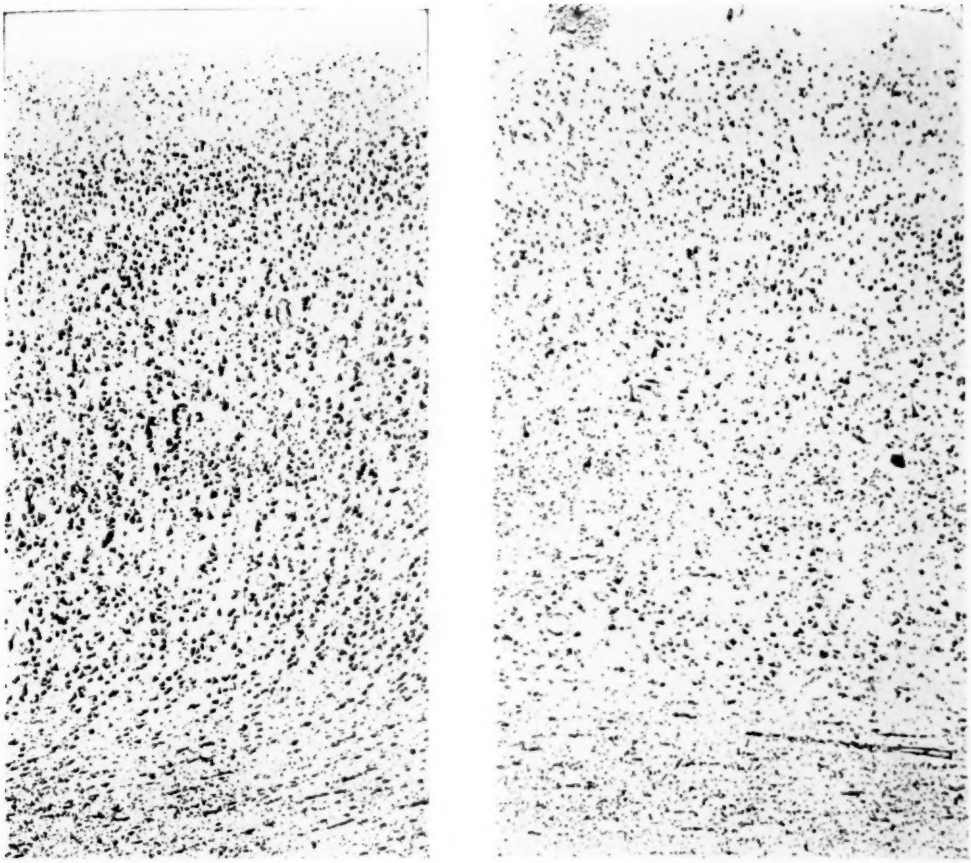


Fig. 1. The frontal cortex in the Nissl preparation. (a) showing normal cytoarchitectonics. (b) showing widespread neurocellular loss, atrophy of single neurons and increase of glia (case 1).

neuroglia was present throughout the frontal lobe. This reaction, however, was not proportional to the degree of cyto-architectural damage, since areas of marked cellular loss could be observed in which scanty glia nuclei were present, while there were numerous glia cells in areas of comparatively minor neuro-cellular damage. Throughout the cortex, small acellular areas were observed containing a detritus which stained slightly metachromatically with thionine. These areas apparently corresponded to the senile plaques as seen in silver preparations. High power examination of the Nissl preparations revealed further details of the neurocellular alterations. There was hardly a normally appearing nerve cell. The majority of the neurons showed the characteristic features of "cellular shrinkage" of Spielmeyer or "chronic cell disease" of Nissl. The cellular body was reduced in size, the apical dendrite showed a corkscrew appearance, the nucleus was dark in color and often could be hardly distinguished from the cytoplasm. This last was darker than normal, showed homogenous structure and often contained a large amount of pigment which stained yellowish or greenish in the thionin preparation. Neuronophagia was very frequent. The increased glia nuclei showed the typical features of a normal glia, degenerative changes such as ameboid alterations were absent.

In the precentral region, the cellular changes, as seen in the Nissl preparation, were of lesser degree. The third layer appeared particularly involved, while the fifth layer was almost intact. Curiously enough, the Betz cells were well preserved throughout, showing normal size and well differentiated tigroid granuli. Only occasionally was pigment found in a moderate amount within the cytoplasm of a Betz cell.

Cyto-architectural study of various fields of the temporal and occipital lobe showed lesions similar to those in the frontal lobe, diffuse rarefaction and at times more pronounced involvement of the third and fifth layers.

The neuron cells of the corpus striatum were considerably decreased in number and the glia had proliferated. The cells of the thalamus and hypothalamic nuclei, the substantia nigra and the red nucleus were much better preserved than the cells of the cortex and the striatum. In the cerebellum, numerous Purkinje cells had

disappeared. The granular layer was thinner than normal. In addition, the cells of the dentate nucleus showed severe changes of the chronic type with compensatory proliferation of the glia. Similar alterations were found in the inferior olives.

In Spielmeyer's preparations for myelin sheaths, the radial and longitudinal fibers in the cortical gray matter appeared diminished in number, and occasionally the myelin sheaths of the subcortical layers were palely stained and thinner than normal.

In addition to this process of atrophy of the cortex, an extremely large number of senile plaques and Alzheimer's neurofibrillary changes of nerve cells was present. The senile plaques were found mainly in the gray matter of the cortex (Fig. 2) and the striatum. Occasionally, a few plaques were present in the white matter, particularly in the subcortical layer. In the cortex, they were evenly distributed throughout the laminae, with the exception of the first lamina where they were rare. The plaques were predominant in the frontal and temporal lobes. To a lesser degree they were present in the occipital and parietal cortex. In the corpus striatum, they were extremely numerous and very small, forming a dust-like argentophilic precipitate. In the cerebellum, the medulla, pons, midbrain and thalamus, the plaques were rare. The morphological characteristics of the plaques varied considerably, from minute plaques which were made up of argentophile, granuli and interwoven fibers, to large plaques with a nuclear-like body of amorphous structure surrounded by a pale ring of fragmented fibrils and by a dark granular crown at the periphery. The astrocytes surrounding the plaques showed hypertrophic and degenerative changes. Hypertrophic and partly degenerated microglia was often seen surrounding the plaques. Microgliacytes were also found within a plaque where they occasionally constituted its nuclear-like portion (Fig. 3). The microglia cells exhibited swelling and vacuolization of the plasma, alterations of the nucleus, uneven argentophilia, knob-like terminations and fragmentation of the processes. Very often these degenerative changes were so advanced as to make it difficult to recognize the morphology of the cells.

Alzheimer neurofibrillary degenerations of the nerve cells were most prominent in the frontal and parietal lobes, Ammon's horn

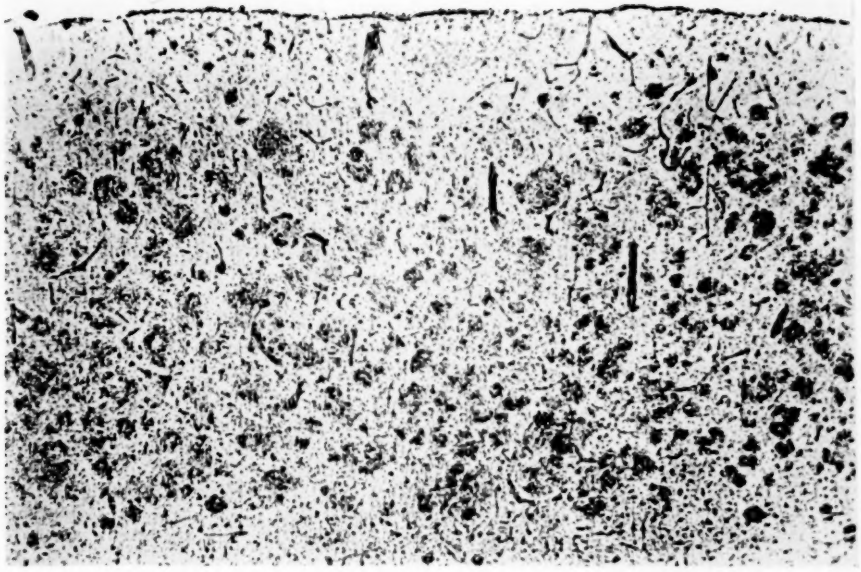


Fig. 2. The frontal cortex in the silver preparation (Braunmühl's method) showing tremendous amount of senile plaques. Low power.

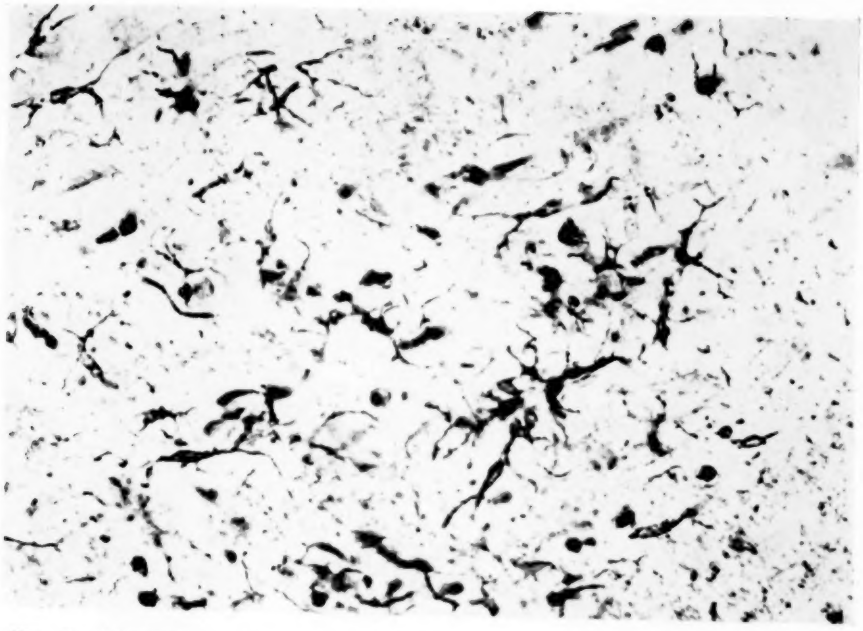


Fig. 3. Microglia reaction about the senile plaques showing hypertrophic and degenerated microgliacytes. Hortega's stain. High power.

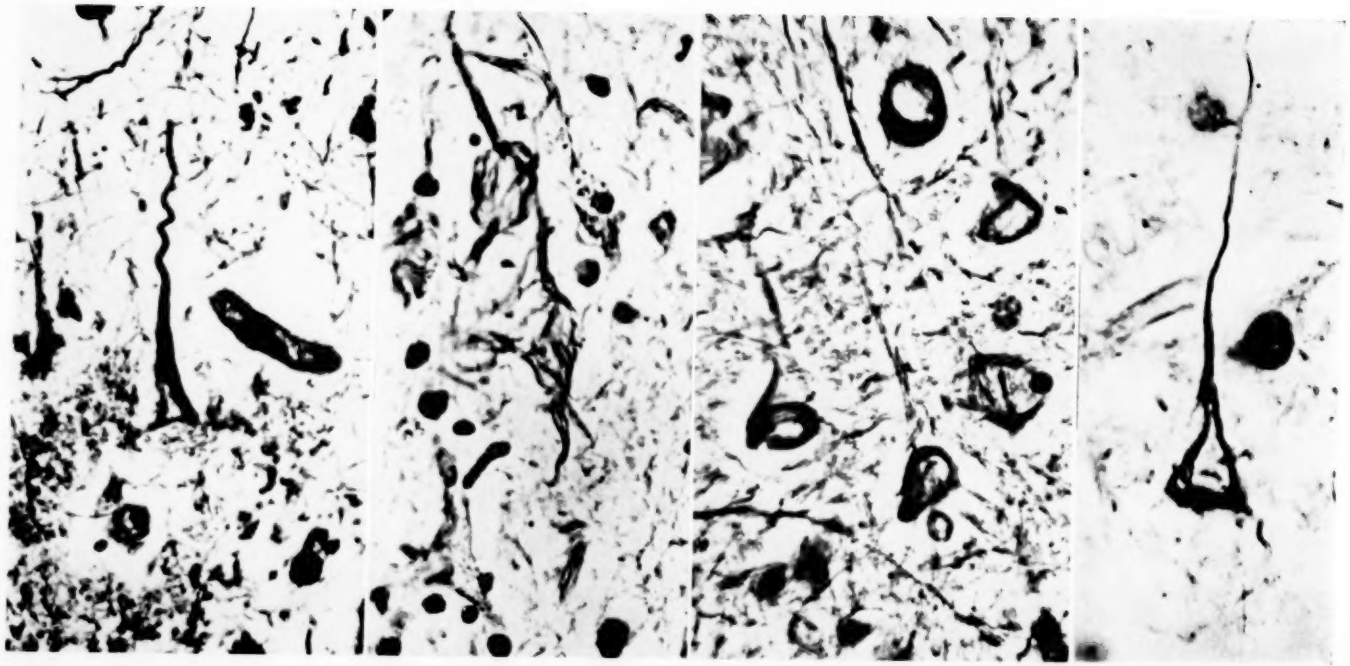


Fig. 4. High power view of Alzheimer's neurofibrillary changes. Silver stain.

and to a lesser extent in the other cortical regions (Fig. 4). They were practically absent in the basal ganglia, pons, midbrain, cerebellum and medulla. The pyramidal cells of the third and fifth cortical layers appeared more frequently involved than other cells. In the regions most involved, the proportion of diseased to normal cells was 1 to 5 or 6, while in other regions this type of alteration was not frequent. There was no correlation between certain cyto-architectural areas and frequency of Alzheimer's changes. In fact, the latter were scattered at random throughout the cortex. All stages of neurofibrillary disease could be detected from a thick and dense neurofibrillar network displaced on one side of the cell to the well-known oval, comma, spiral and basket-like appearances.

Case 2. This case will be only briefly mentioned since it has already formed the subject of a publication from this laboratory.[3] It concerns a patient, a white male, whose first symptoms became manifest at the age of 38 years, when it was noticed that he became distractible, irritable, impulsive and forgetful. A year later, following an undetermined attack in the course of which he was semiconscious, the patient became more confused and depressed, disclosed gross memory defect, became more restless and unmanageable. The mental condition became progressively worse and the patient was committed in July, 1928.

At physical examination, the patient showed slow and indistinct speech, impairment in writing and apraxia. At mental examination, he appeared indifferent and seclusive. His memory showed most defect in recent rather than remote events. His retention and immediate recall were poor. He could not calculate. General everyday knowledge was deficient. Insight and judgment were extremely poor. The ability to concentrate and to focus attention was greatly altered. About a year later, speech was more defective and slurred. There were slowness and awkwardness in walking; some incoordination of both lower extremities, increased tonicity of the muscles; hyperreflexia with bilateral patellar and ankle clonus, diminished abdominal and cremasteric reflexes. Further determination of the mental status revealed a rapid and progressive deterioration. He was dull, indifferent and careless about his personal habits. He was limited in his understanding. Questions were answered mostly in a mumbling and unintelligible manner.

He gave an impression of emotional disintegration, apathy alternating with periods of slight euphoria. He was completely disoriented in all spheres. He gradually became bedridden and incontinent of urine and feces. Occasional twitchings of the body were noticed with extreme rigidity. He continued in this vegetative condition and died November 6, 1929, of bronchopneumonia.

At postmortem, the brain was grossly smaller than normal. The convolutions were diffusely atrophic. There was no gross evidence of cerebral arteriosclerosis. Sections of different cortical areas, stained with Nissl's method showed marked nerve cell changes consisting of a diffuse diminution of cortical nerve cells due to various degenerative changes, chiefly shrinkage and sclerosis. Senile plaques were extremely numerous throughout the cortex and the corpus striatum. Both astrocytes and microglia surrounding the plaques showed signs of intense reaction, exhibiting hypertrophic and degenerative alterations. The Alzheimer neurofibrillary degeneration of the nerve cells was also frequently found, being most prominent in the frontal and parietal lobe, the Ammon's horn and to a lesser extent in the other cortical regions. There was no histological evidence of arteriosclerotic changes of the blood vessels.

Comment

There can be little doubt that the two cases here presented are typical instances of Alzheimer's disease. All clinical details but the age of onset, are in accordance with the classical description of the disease. In both cases, the mental reaction was organic in type and the course was progressive and rapid. Speech defects and aphasia were present. Neurologic signs such as anisocoria, overactivity of deep reflexes, muscular hypertonicity and involuntary movements were likewise observed in both cases. The occurrence of general convulsion completed the clinical picture. From a pathologic point of view both cases could be considered also as typical examples of Alzheimer's disease, the classical pathologic picture being present in all its features.

A critical survey of the literature discloses a few other cases in which all clinical and pathologic details but the age of onset are in accordance with the description of Alzheimer's disease.

The first case was described by Perusini[4] in 1909. It concerned a male who at 39 years of age showed personality changes, marked memory impairment and emotional instability. In addition, aphasia, agraphia, alexia, dysarthria were observed, together with motor incoordination, tremors and epileptic attacks. Severe dementia developed early and the patient died at 46 years of age. Pathological examination disclosed diffuse atrophy of the cortex and a large number of senile plaques and neurofibrillary alterations.

A second case, described by Urechia and Danetz[5] in 1924, concerns a female patient age 37 at the time of onset of the first symptoms. These consisted of memory loss, disinterest, convulsions, apraxia and paraphasia. There were also rigidity and spastic contractures. The patient died at 48 years of age. Histopathological examination revealed the usual features of Alzheimer's disease, i. e., cerebral atrophy, senile plaques and Alzheimer's neurofibrillary disease.

A further case illustrated by Schottky[6] in 1932 refers to a female patient who at 38 years of age showed lack of initiative, memory impairment and emotional instability. Some time later, her speech became slow and she gradually developed a bilateral apraxia and agraphia. As the intellectual impairment became more serious, definite logoclonia and paraphasia became evident. There was a marked disturbance in gait with spastic contractures and facial weakness. The patient died at 45 years. Pathologically, the characteristic changes of Alzheimer's disease were observed.

A fourth case was described by Lowenberg and Waggoner.[1c] A male, aged 34, presented gradual mental deterioration with episodes on excitement over a period of three years. The examination showed intellectual impairment, memory disturbances and confusion. In addition, neurological signs were present consisting of hyperactivity of the reflexes, pyramidal tract involvement and mild incoordination. General convulsions occurred twice during the course of the disease which lasted about six years. Pathologically, countless senile plaques were diffusely present in the cortex, basal ganglia, cerebellum and spinal cord. Alzheimer's neurofibrillary changes were frequently found in the cerebral cortex.

Finally, a fifth case was recently described by Kufs.[7] It con-

cerns a male patient who, at the age of 28 years, showed some memory impairment. There was little change for some three years at the end of which period, more marked mental changes were observed. These consisted of a gradual mental deterioration coexisting with speech disorders. Epileptic seizures and choreo-athetosis developed at a later age. Death occurred at 39 years. In addition to the usual findings of senile plaques and neurofibrillary changes, there was marked atrophy of the basal ganglia and sclerosis of the inferior olives.

A few other early cases of Alzheimer's disease have been reported. These disclose, however, either from the clinical or from the pathological point of view considerable variations and should be considered as doubtful instances of the disease.

It is, in fact, necessary that both clinical and pathological features corresponding to the original description be present in order to establish the diagnosis of Alzheimer's disease, and since pathologic findings of senile plaques and fibrillary changes are by no means indicative of the disease,[3] the clinical features and the course are of essential importance.

The first of these doubtful cases was described by Schnitzler[8] in 1911. A female patient showed the first mental symptoms at the age of 32 and died at the age of 37, of a slowly progressive apathetic dementia. Definite cortical focal signs such as aphasia and apraxia were not present. Transient neurological features such as facial weakness, ptosis, bulbar signs, paresis of left fingers appeared during the last period of the disease. Myxoedema was present. Pathologically, there were neurofibrillary changes but no senile plaques. Since neurofibrillary changes have been reported in experimentally thyroidectomized animals (Balli,[9] Rasdolsky[10]) there is the distinct possibility that myxoedema was responsible for the neurofibrillary pathology.

The second case, described by Barrett[11] in 1913, refers to a female, age 35, who clinically exhibited gradual mental deterioration with restlessness, disorientation, euphoria, alternating with depressive moods. Generalized convulsions occurred frequently. The important neurological feature which casts doubt as to the nature of the disease was represented by considerable muscular atrophies and paresis coexisting with rigidity, and bilateral increases of the

deep reflexes as seen in lateral sclerosis. Pathologically, there was marked degeneration of the pyramidal tract. Senile plaques and neurofibrillary disease were present. The fact, however, that these alterations may occur in cases of amyotrophic lateral sclerosis[12] casts considerable doubt on the significance of these findings in Barrett's case.

An additional case, described by Weimann[13] refers to a male, aged 37, in whom the mental features were those of behavior disturbances, compulsions and restlessness. Focal neurological signs were scanty. The clinical data are too atypical, as Kraepelin[14] himself pointed out, to justify the diagnosis of Alzheimer's disease. Pathologically, neurofibrillary changes were found but no senile plaques. In addition, there were marked deposits of calcium in the nervous system and advanced sclerosis of the vessels of the brain.

Including the two cases here presented, there are, in conclusion, seven instances of typical Alzheimer's disease with onset before the presenium and three further doubtful cases. All these cases have been included in the "juvenile type" of Alzheimer's disease. This term appears, however, hardly justified. In fact only in one of all instances reported under this heading, did the symptoms occur in the juvenile age. This case was that of a mentally defective boy, reported by Malamud and Lowenberg,[1c] who at 15 years of age showed excitement, confusion, compulsion and negativism. No neurologic symptoms and particularly no focal cortical signs were present. There was a remission of about four years in the course of the disease which lasted four years. Although the pathology was that of Alzheimer's disease, the uncharacteristic mental picture, the absence of cortical focal signs and the long remission leave much doubt as to the classification of this unusual case.

It will be noted that, with this exception, all cases reported in the literature developed in the adult age between the twenty-fifth and fortieth year. It appears, therefore, that the term "adult type" of Alzheimer's disease is more appropriate than "juvenile type."

In this attempt at establishing this adult variety, it seems safe to leave a borderline period of a few years before the onset of the presenium, generally considered at 45 years. This period may be arbitrarily fixed between 40 and 45 years. Therefore, cases with

onset after 40 years of age would be considered as of the classic presenile type; cases developing in the third and fourth decade of life would be included in the "adult type;" cases developing before the 20s could be classified as "juvenile type." The establishment of this last type, however, is not yet justified by actual clinico-pathologic material.

A further attempt to subdivide Alzheimer's disease into clinical types has been made by Lowenberg and Waggoner[1c] who distinguished an intermediary type occurring shortly before the presenium in which heredity seems to play a determined rôle. However, additional cases have been reported (Grünthal and Wenger,[15] McMenemy, et al.[16]) in which although familial incidence was present, the age of the patients fell within the presenium. More evidence is needed, therefore, to support Lowenberg and Waggoner's contention.

While of doubtless interest from a clinical point of view, the subdivision of Alzheimer's disease into several types has a far-reaching importance in the study of the causation of the disease. To be sure, any discussion as to whether Alzheimer's disease is closely related to or independent of senility, is of little significance if the nature of the process of brain senescence is not adequately determined. Of the various theories on senescence, the one recently brought forward by Braunmühl[17] appears to offer interesting clues for the problem under discussion. According to this authority who bases his conclusions upon physico-chemical laws, aging of the brain is essentially a manifestation of primary changes of the unstable brain colloids from a highly dispersed to a less dispersed condition, eventually resulting in condensation and coagulation. This process is known as "syneresis" and is highly characteristic of the aging of any colloidal system. The formation of senile plaques and neurofibrillary changes is a secondary manifestation of these colloidal changes. In Braunmühl's words, "What is called syneretic process in colloidal chemistry is equivalent to atrophic process in neuropathology."

Basing on this theory, it is conceivable that "syneretic" mechanisms which operate during the physiologic aging, may act at an earlier age and with particular intensity, upon colloidal systems constitutionally predisposed. It will be noted that in case 1 con-

stitutional familial factors can be surmised since two siblings were affected by mental diseases. Additional factors are likely to play a rôle in the causation of early colloidal changes. Among others, vascular mechanisms should be kept in mind. It is known, in fact, that in cases of widespread brain arteriosclerosis extreme cortical atrophy may be found, due to diffuse interference with the blood supply of the cortex. To be sure, morphologic changes of the brain blood vessels are usually absent in Alzheimer's disease, but one can reasonably conceive that transitory impairment of the cerebral circulation due to vasomotor disturbances may have some rôle in the interplay of various factors bringing about the pathologic changes.

It appears, in conclusion, that a better knowledge of the nature of both presenile and adult types of Alzheimer's disease may be expected from the study of the causes and the mechanism of the physico-chemical alterations occurring in the aging brain.

Acknowledgment

The authors are indebted to Dr. W. W. Wright, superintendent of Marcy State Hospital, for the pathologic material and clinical notes, and to Dr. H. A. Steckel, director of the Syracuse Psychopathic Hospital, for information concerning the first stage of the disease in case 1.

Department of Neuropathology
New York State Psychiatric Institute and Hospital
New York, N. Y.

REFERENCES

1. a. Malamud, W., and Lowenberg, K.: Alzheimer's disease. A contribution to its etiology and classification. Arch. of Neur. and Psychiat., 21:805, 1929.
   b. Lowenberg, K., and Rothschild, D.: Alzheimer's disease: its occurrence on the basis of a variety of etiologic factors. Am. J. Psychiat., 11:269, 1931.
   c. Lowenberg, K., and Waggoner, R. W.: Familial organic psychosis (Alzheimer's type). Arch. of Neur. and Psychiat., 31:737, 1934.
2. Braunmühl, A.: Die psychischen Störungen des Rückbildungalters. Anatomischer Teil. Ztsch. f. ges. Neur. und Psychiat., 167:78, 1939.
3. Jervis, G. A., and Soltz, S. E.: Alzheimer's disease—The so-called juvenile type. Am. J. Psychiat., 93:39, 1936.
4. Perusini, G.: Über klinische u. histologische eigenartige psychische Erkrankungen des späteren Lebenalters. Nissl-Alzheimer Arb. 3:297, 1910.
5. Urechia, C. I., and Danetz, C.: Quelques Considerations sur la Maladie d'Alzheimer. Encephale., 19:382, 1924.

6. Schottky, J.: Über präsenile Verblödungen. Ztsch. f. ges. Neur. und Psychiat., 140:333, 1932.

7. Kufs, T.: Über eine Frühfrom der Alzheimerschen Krankheit mit Olivensklerose und hochgradiger Atrophie der Stammganglien. Arch. f. Psychiat., 107:431, 1937.

8. Schnitzler, J. G.: Zur Abgrenzung der sog. Alzheimerschen Krankheit. Ztsch. f. ges. Neur. und Psychiat., 7:34, 1911.

9. Balli, R.: Lesioni des Reticolo-Neurofibrillare Endocellulare in Mammiferi Adulti Privati dell'Apparecchio Tiro-paratiroideo. Riv. Sper. Freniat., 32:803, 1907.

10. Rasdolsky, J.: Histologische Veränderungen in dem zentralen u. peripherischen Nervensystem der Tiere mit extirpierten Schild-u. Nebenschilddrüsen. Ztschr. f. ges. Neur. und Psychiat., 106:96, 1926.

11. Barrett, A. M.: A case of Alzheimer's disease with unusual neurological disturbances. Jour. of Nerv. and Ment. Dis., 40:361, 1913.

12. Bogaert, D., and Bertrand, I.: Pathologic changes of senile type in Charcot's disease. Arch. of Neurol. and Psychiat., 16:263, 1926.

13. Weimann, W.: Über einen atypischen, präsenilen Verblödungprozess. Zentbl. Neurol., 23: 355, 1921.

14. Kraepelin, E.: In discussion of Weimann's paper (See 13).

15. Grünthal, E., and Wenger, O.: Nachweis von Erblichkeit bei Alzheimerscher Krankheit nebst Bemerkungen über den Altersvorgang im Gehirn. Monatsch. f. Psychiat. u. Neurol., 101:8, 1939.

16. McMenemy, W. H., Worster-Drought, C., Flind, J., and Williams, H. C.: Familial presenile dementia. Jour. of Neurol. and Psychiat., 2:293, 1939.

17. a. Braunmühl, A.: Kolloidchemische Betrachtungsweise seniler und präseniler Gewebsveränderungen. Ztsch. f. Neurol. u. Psychiat., 142:1, 1932.

b. Braunmühl, A.: Versuche um eine colloidchemische Pathologie des Zentralnervensystems. Klin. Wochenschr., 13:897, 1934.

COMPARISON OF NONPSYCHOTIC WOMEN WITH SCHIZOPHRENICS WITH RESPECT TO BODY TYPE, SIGNS OF AUTONOMIC IMBALANCE AND MENSTRUAL HISTORY*

BY HELEN E. ELLIOTT, M. D.

Inasmuch as the opportunity presented itself to examine a number of nonpsychotic young women in the same age group (15-35 years) as the majority of recently admitted cases of schizophrenia, curiosity prompted the use of 100 of this group as controls for comparison with an equal number of women patients.

The mean age for the control group was 26½ years, that of the schizophrenic group, 27 years. Only objective, readily obtainable signs were checked, as it was not considered feasible to subject the control group to pharmacological tests.

The data so collected divided themselves into three sections: (1) incidence of occurrence of different body types, (2) presence or absence of signs of autonomic imbalance, and (3) variations in menstrual histories.

With respect to the characteristics typifying the various physiques as observed in women, Kretschmer[1] states that the attributes are more indefinite and atypical in this sex, and in addition he points out variations from the male. Thus in asthenic women, a degree of hypoplasia is expected, the average height in his studies being just over five feet. Further, the deficiency in thickness, found in the male coupled with unlessened length, is often present as part of a general underdevelopment. Occurrence of an angular profile and a short chin intensifies the type; however, heterogeneous face shapes were often found by him in this group. The athletic type in women follows the male formulation with certain characteristic deviations. The figure may vary from a well-rounded one to one with outstanding musculature. The fat development is distinctly proportional to the bony structure. One looks also for prominent malar eminences; a broad midface occurs as well as the steep-egg shaped which is expected in the male. A diathesis toward acne is often present.

*Read before the interhospital conference held at the Utica State Hospital, Utica, N. Y., April 27, 1940.

In classifying the pyknic type, Kretschmer avers that in hard workers and in young people (and although he does not, one may well include devotees of the present fad for slenderness), the expected compact overlay of fat is absent. Hence the basis of determination lies in the findings of depression of the shoulder inner deltoid curve, small bones, a smooth-contoured, five-cornered face (frequently delicate in impression), and a short, thick neck.

On the subject of the diagnostic significance of hair growth among women, Kretschmer states he has not discovered any fundamental difference in the distribution on trunk, head or extremities, it being in general similar to the male and in the asthenic scanty with full eyebrows often joining over the nose. As a rule the hair of pubis and armpits in pyknics is coarse and strong. The classification of the selected women according to physique followed the prototypes just briefly described.

TABLE 1

	Asthenic	Athletic	Pyknic	Dysplastic	Undetermined
Nonpsychotic group	25	31	40	2	2
Schizophrenic group:					
Simple type	1	0	1	..	..
Catatonic	10	11	2	..	..
Paranoid	21	8	4	2	..
Hebephrenic	17	17	3	1	2
Total	49	36	10	3	2

The predominance of the first two types in the schizophrenic group is self-evident, illustrating the correlation between body build and temperament postulated by Kretschmer; this is reenforced by the variation in occurrence in the two groups. Incidentally, while the majority of the women in the nonpsychotic group appeared superficially well adjusted, seven evinced stigmata of instability, with untoward emotional responses; one admitted hospitalization for "nervous breakdown;" one was negativistic and admitted seclusiveness and feelings of inferiority; four of these women were distinctly asthenic, the other athletic in habitus.

A comparison of deviations from the normal in hair distribution revealed nothing significant. The type of hair found in the pyknic women bore out Kretschmer's conclusions.

TABLE 2. MALE HAIR DISTRIBUTION

	Pubic	Facial	Body
Nonpsychotic group	22	2	1
Schizophrenic group	10	5	2

Before presenting the findings relating to the functioning of the autonomic nervous system in these groups, it may be advisable to review some of the concepts involved.

The vegetative or autonomic nervous system embraces the parasympathetic or extended vagus, that is the craniosacral division and the sympathetic or thoracicolumbar ganglia. These groups of ganglia, through double innervation of all vegetative organs, have antagonistic effect one upon the other resulting normally in physiological balance. Ascendancy of one over the other results in the syndromes, so-called, of vagotonia or sympathicotonia. As Wechsler[2] states, experience shows that sharp division does not exist; this is confirmed by Bernhardt,[3] who found that preponderance may occur in but one system or group of organs, the leading one in determination being the cardiovascular. In fact other organs may show a contrary condition. Excessive vagal activity produces bradycardia, hypotension, pronounced sinus arrhythmia, a tendency to spasticity of the gut with easy vomiting, urticaria, dermatographia, clammy hands and feet, miosis, narrowing of the palpebral fissure, and in women a tendency to a male pelvic hair line and a ring of hair around the nipples. In addition vagotonics often display an active Aschner ocular phenomenon (slowing of the pulse when pressure is applied to eyeballs). In sympathicotonia one finds an absent or diminished oculocardiac reflex, dilated, mobile pupils, dry warm skin, a tendency to acceleration of the pulse and hypertension. An active ciliospinal reflex is expected. In youth some degree of vagotonia is physiologic and in advancing age the sympathetic system gains in ascendancy. Simpson[4] includes among the symptoms of vagotonia, acne, ease of fatigue and lack of confidence; he attributes to sympathicotonia premature grayness of the hair and psychic flushing.

Ever since Eppinger and Hess[5] in 1909 postulated vagotonia and sympathicotonia as clinical entities and claimed to have found the former predominating in schizophrenia, various investigators have studied groups of schizophrenic patients in attempts to substantiate these premises. On the contrary, recent reports point to the

existence of a neurovegetative anergy, a diminished function of the vegetative nervous system. This is the conclusion of Ancochea and Cuevillas.[6] Freeman and Carmichael,[7] using adrenalin injections and considering variations in blood pressure and heart rate as criteria, studied both normal and schizophrenic individuals and claim that a diminished reactivity was found in the latter, but admit that in some cases a slowing of the pulse occurred which might be attributed to a strong vagal tonus. The findings in the cases herein examined are inconclusive.

TABLE 3

	Nonpsychotic	Psychotic
Vagotonic symptoms		
Sinus arrhythmia	35	34
Positive Aschner test	9	22
Hypotension	9	14
Dermatographia	19	31
Acne	17	40
Nipple hair line	17	17
Spastic colon	5	9
Cold extremities	24	39
Cyanosis of extremities	0	4
Miosis	2	10
Sympathetic symptoms		
Tachycardia	0	12
Hypertension	2	4
Active ciliospinal reflex	59	14
Mydriasis	39	29

The preponderance of dermatographia, acne, cold clamminess of extremities and more frequent response to Aschner test might signify an increased vagal tonus in the patients. This is borne out by the less active sympathetic reaction as shown in lessened response to ciliospinal reflex—a normal reaction. Five of the control group and 15 of the psychotic women exhibited the presence of a combination of three or more symptoms of autonomic imbalance. This incidence is not sufficient to be significant, but one may suggest that it shows a trend. (It may be noted also that no correlation between body type and signs of autonomic imbalance seems to exist.)

During the examination of the pupils on light stimulation, the incidence of hippus, i. e., alternate contraction and dilatation, was found to be 10 in the controls and 15 in the patient group. The significance of this occurrence is not known, but it is noteworthy that in every instance it appeared in conjunction with some evidence of autonomic imbalance.

In reporting any data regarding menstrual function, one is necessarily confronted by the problem of defining terms. The menstrual cycles as reported by Latz and Reiner[8] show that every woman is a law unto herself, that 90 per cent in an apparently normal group showed variations of from two to eight days. Rock and Bartlett[9] describe as normal menstruation uterine bleeding in cycles not shorter than 24 or longer than 31 days, and lasting at least two days. These criteria have been adopted in the terminology used in this recording in which the menstrual histories of the 100 controls are contrasted with those of 69 of the schizophrenics. The latter number is limited to those where statements of patients or the accounts of relatives could be regarded as reliable. On this basis 77 of the nonpsychotics would be in the normal range; of those displaying aperidomenorrhea, 12 exhibited oligomenorrhea of whom two had a regular cycle of 40 days; the others varied from four days to two weeks irregularity. Eleven gave a history of polymenorrhea. In the psychotic group's prehospital history, only 45 could be classified as having normal menstruation, 17 oligomenorrhea and 7 polymenorrhea. Thus it would appear that some disturbance of menstrual function was present prepsychotically in an appreciable number of the schizophrenic women. In this connection it is interesting to note that the age of onset of menstruation varied widely in the latter group: 70 per cent of the controls began menstruation in their twelfth, thirteenth, or fourteenth years, but only 60 per cent of the schizophrenics did likewise; the hebephrenics showed the greatest scattering (from 10 to 18 years).

Except for a reaffirming of Kretschmer's conception of the correlation between physiques and types of mental disorder, nothing

definite has been ascertained by this study. Nevertheless the findings appear to signify, by the greater number of symptoms of autonomic imbalance and of aperidomenorrhea in the psychotic group than in the nonpsychotic group, that the soma does not function as efficiently nor in as well-balanced a manner in schizophrenic women.

Binghamton State Hospital
Binghamton, N. Y.

REFERENCES

1. Kretschmer, E.: Physique and Character. Harcourt, Brace and Company, New York, 1925.
2. Wechsler, Israel S.: Textbook of Clinical Neurology. Third edition. The W. B. Saunders Company, Philadelphia and London, 1937.
3. Bernhardt, H.: How may one recognize a sympathicotonic and a vagotonic patient? Zeitschr. für arztl. Fortbild., 35:202, 1928.
4. Simpson, Virgil: Thyrotoxicosis and associated vagotonic and sympathicotonic syndromes. Am. Jour. Surg., 3:249, 1927.
5. Eppinger, H., and Hess, Leo: Vagotonia. Nervous and Mental Disease Publishing Co., New York. 1917.
6. Ancochea, N., and Cuevillas, R. D.: Tests of vegetative nervous system in schizophrenia. Arch. de Méd. Chir., 37:1103, 1934.
7. Freeman, H., and Carmichael, H. T.: The autonomic nervous system in schizophrenia. Arch. Neurol. and Psychiat., 33:342, 1935.
8. Latz, Leo J., and Reiner, E.: Natural conceptions. J. A. M. A., 105:16, 1935.
9. Rock, J., and Bartlett, M. K.: Biopsy studies of human endometrium. J. A. M. A., 108:24, 1937.

PARANOID REACTIONS IN THREE GENERATIONS*

BY H. G. RAINEY, M. D., AND WILLIAM R. CARSON, M. D.

The purpose of this presentation is not to discuss heredity in mental disease, but merely to present case reports of patients of three generations admitted to St. Lawrence State Hospital. These patients all exhibited paranoid reactions.

It is not felt that any conclusions can be drawn, and the reports are made only for the interest attached to them.

In Meyerson's[1] study he was able to find only nine instances of three-generation psychoses admitted to the Taunton State Hospital, although he reports an added group of 11 three-generation psychoses in which one of the generations was never admitted to a State hospital but in which he feels that the presence of a psychosis was definitely demonstrable from the history. In none of his cases and in no other instance have the writers been able to find three-generation psychoses admitted to the Taunton State Hospitalized.

It is felt that this series is unusual in that both parents of the first generation were admitted to the hospital.

Case 1. (*John Doe*) *First Generation*

Family History: The patient's father was an alcoholic; he was nervous and irritable. The uncles, aunts, sisters and brothers were considered to be of a "nervous" temperament.

Personal History: The patient was 49, born in Ireland. He attended school until about nine, worked at gardening all his life. He married in 1884 and his married life was considered to be congenial; five children resulted. Following an injury in 1906, about one year prior to his admission, his right side had been weak and he exhibited a speech difficulty. He was admitted to the hospital on a petition stating: "At times grows excited without provocation; claims his family is against him; is irritable and despondent; wants to go to the poorhouse; leaves his home and stays away without notifying any one of his whereabouts." The patient himself stated in the examiners' presence: "The air paralyzes him when it

*Read before the interhospital conference held at the Utica State Hospital, Utica, N. Y., April 27, 1940.

strikes his body; he has to feel himself to know if he is there and alive; his family do not treat him well and give him good care; said he heard voices calling to him and that his head vibrated."

On admission to the hospital, the patient showed some depression and a paranoid trend directed against members of his family. He complained that they were against him, that they showed him no respect, and that his wife tried to injure him with his employers. A diagnosis was made of simple depression with mild paranoid condition. After his admission, he expressed further paranoid ideas against his wife, the nurses, and the attendants. He asserted that his wife had told his children to like her better than himself, and he also said that nurses and attendants turned air upon him and that this air was paralyzing and killing him. His stay at the hospital was short. He had pernicious anemia which ran a rapid course, and he died on October 17, 1907. Physical examination upon admission to the hospital had demonstrated the presence of the anemia.

Case 2. (*Mary, Wife of John Doe*) *First Generation*

Information was obtained from two daughters of the patient. One was reluctant to give information to anyone but the superintendent or assistant superintendent. She was very "nervous," objected to her mother being in the hospital, and cried on several occasions.

Family History: There was no history of nervousness, mental disorder or epilepsy in the family.

Personal History: The patient was 62, and was born in Ireland. The only available information of her early life showed that she attended school until about 14 and was married at 19. Marriage for her, as well as for her husband, was considered to be very congenial. The patient was described as of a domineering disposition with a "do-or-die" attitude. She was said to be cheerful, a good mixer, a good manager and an excellent housekeeper. At the age of 48, she suffered from what was considered to be her first "nervous breakdown." She became highly overactive and overtalkative, imagining that everyone in the house, including herself, had tuberculosis. Her physicians advised a complete change, and she went to California where she remained about nine months.

Nothing is available to indicate what occurred during this period. Upon her return she was exceedingly irritable; occasionally she would become enraged at the daughter with whom she lived and would not speak to her for weeks at a time. In 1919 she became highly interested in politics and quarreled with anyone who disagreed with her in this field. For five years she refused to go into the homes of any of her former friends, giving as her reason these people's "betrayal" of the Irish cause in which she herself was interested. She consulted a physician in 1925 because of bladder trouble. He apparently treated her locally and gave her some green pills. The patient asserted that the doctor poisoned her because of their difference in opinion on the Irish question. She consulted another doctor and in time turned against him also. This doctor's nurse was giving her serum; the patient would not return for further treatment, saying the nurse was not giving the serum correctly. She went to two hospitals, where she was treated for cystitis. One hospital requested her to leave, the authorities saying she was "a mean old lady." She developed ideas against the daughter, accusing her of hiring doctors to persecute her. She told the daughter frequently that she would be destroyed and justice would be performed. Finally she threatened to drown herself and was committed to the hospital.

On admission she expressed a well-fixed, systematized trend directed principally against doctors. She said one doctor had placed a painful foreign body in her bladder because of difference in political opinion. She did not say that the doctor wished her to die, but the inference was obtained from her that he would be perfectly willing to have her do so. She declared that this doctor was not a fit man to practise medicine; that she had reported him to the district attorney and had requested his removal by the governor from his position. She said, however, she would not take the law into her own hands by doing physical violence.

The patient has since remained at the hospital, her condition essentially unchanged. She contends that she is being held illegally, and she tells other patients they should not engage in certain forms of occupational therapy, and that they should write to their families and insist on being removed from the hospital.

This woman has written letters to mayors, district attorneys and to judges of various cities, to the governor and to Pope Pius XI. She has written also to Samuel Hoare, Anthony Eden and Neville Chamberlain, telling them that they have been unjust and that there are people ready to "smash" them at any moment. In all these letters she asserts that she is being discriminated against and is being held illegally; and she demands her freedom. Her letters also go to relatives of other patients reporting what she considers to be unfair treatment of patients in the hospital. There have been spells in which she would become highly irritable, threatening and assaultive. Paranoid delusions have continued, including the belief that her food was poisoned. She has expressed ideas of outside influence, saying that other patients are interfering with her and that they do this by means of electricity. A diagnosis of paranoia was made.

Physical examination upon admission showed her to be fairly nourished, well developed. Her respiratory system was normal. The cardiovascular system showed some slight edema of the lower extremity. Her blood pressure was 114/68, hemoglobin 80 per cent. There were no subjective complaints.

Case 3. (*Jane, Daughter of John and Mary Doe and Wife of Richard Roe*) *Second Generation*

Information was obtained from the husband and the sister of the patient. According to the anamnesis, the sister was rather "nervous" and it was difficult to get facts from her.

Family History: Both parents were patients at the St. Lawrence State Hospital (preceding case reports). The patient had three sisters and one brother; the brother was said to have had "nervous trouble" but never had been in an institution.

Personal History: Born in New York State, the patient was the third child. Her mother is said to have been well during the prenatal period and the birth was normal. The baby was breast-fed, and was described as unusually bright and active, walking and talking at the usual age. The little girl had some of the childhood exanthemata, but never had been seriously ill. At six, she started school and learned easily. Descriptions of her school days call her a "good mixer" and a "born leader," a person who "had to be at

the head or not at all." Following graduation from a State normal school, she taught school for four years, taking charge of an art studio during the summers. In all her work she was successful. At 25, she married a professional man, and their married life is described as congenial and happy; and there were three children. It is stated, however, that for 11 years prior to admission she had been "nervous" and had suffered from various physical complaints, with her "nervousness" ascribed to overwork. Her description is that of a "very religious" person, modest, bright, cheerful, slow to anger but hypersensitive, often taking offense when none was intended. It was said that she rarely held a grudge but became upset if anything out of the ordinary happened and that she was inclined to be egotistical and opinionated and to give offense herself because of this. Music interested her greatly. Her hobby seemed to be leading in public affairs. Described as never over-particular about her dress, she was a poor housekeeper but a good mother. If she did not get her own way at all times, she was inclined to sulk.

About four months previous to admission, the patient visited her mother who was then in the State hospital. Following this, she seemed to fail gradually, began to worry about the children, thinking that something was going to happen to them; said her mother had denounced her; began to talk of strong minds working on her mind, disciplining her; said that Mrs. Stillman had been notified by some of her neighbors to foist an Indian baby upon her to complete her disgrace. Three months before her admission, she told her husband that she had "the whole thing solved;" that last year she had made a remark at a certain governor's home that he did not like and that the governor had started a conspiracy to break her husband's political career. Taken to a local hospital, she was overactive and overtalkative, speaking continually of a political conspiracy and of her "immorality;" saying everyone was against her, even her best friends; saying the neighbors were talking about her being intimate with her gardener. The neighbors, she thought, were flashing lights on her.

Two weeks before admission, she thought she heard a strange noise and saw lights flashing on a Lysol bottle. As these lights flashed, something told her to drink the Lysol. The same day she

had callers who spoke about the water being deep in the lake. After they left she thought it was a suggestion for her to drown herself. The next day she drank Lysol, telling her husband that all her friends were around the house blowing their automobile horns. She refused to let people into the house, and it was necessary to enter by a ladder. Taken to the hospital, she received emergency treatment. Accusing people of doping her, she said her friends were organized in a group to break her will.

She entered the hospital applying for voluntary admission. At this time she was suspicious, but cooperative and attentive, although she appeared to think the physician would not believe her story. The trend was characterized by ideas of persecution; but there were no defects in the intellectual field, and she showed some insight. The patient said at times she realized overwork had caused her to think as she did; at other times she said she actually believed her own statements. When she had visited her mother some few months previously, she said, her mother had told her what was going to happen to her. A local doctor had performed an operation on her for repair of the perineum. Her mother had undergone the same operation. Therefore, she now felt as her mother felt. All her trouble had started, she believed, when she made a derogatory remark to a local jurist; she felt this had caused her husband to lose an election for which he was a candidate. Expressing some sexual ideas, she said that on one occasion she talked with a woman concerning each other's marital relations. Immediately following this, she went home and took a douche. She could give no reason for this, but added that at times she thought she was abnormal. She complained of the doctor who repaired her perineum, stating she believed he deliberately harmed her. People thought that she had sexual relations with her gardener, she believed. In admitting the attempt to take her own life, she said she tried suicide because she felt she was a depraved woman and because the light from a hospital nearby kept shining in the bathroom on the Lysol bottle.

Physical examination was essentially negative at the time of admission and all laboratory tests were negative as well. The patient continued to express ideas of persecution, asserting she felt that people had "framed things" so she would have to come to the hos-

pital, and also to injure her husband. The doctor who operated on her, she felt, had worked for her downfall and was her enemy. Within a month after admission, she expressed the belief that this doctor had plotted with the Masons and the Republican Party to ruin her and her husband and children. She showed considerable suspicion of persons at the hospital and began to include the ward physician in her delusional trend, declaring that he had entered into the "plot" to keep her at the hospital. Also she began to blame her husband, and gradually he became one of her "persecutors" rather than one of their other victims. Becoming irritable, she refused to eat. Then she expressed the belief her husband was suffering from a serious disease which she might have given him. The entire staff of the hospital was antagonistic to her, she said, and the ward physician was controlling her children whom he intended to injure. The diagnosis made was that of paranoid condition.

Twenty months after admission she was paroled, although she had shown no improvement. Returned to the hospital within two weeks, she was reparoled three months later. This time she remained out of the hospital for a year and, according to reports from her husband, made a fair adjustment at home, although she continued to express some of the ideas noted in the hospital. Two years and three months after this parole, she was readmitted to the hospital. At that time she was paranoid and admitted renewing her suicide attempts.

According to the interval history, the patient got along well for a few months after she left the hospital. Then she began to worry about having another baby, became suspicious of people, thought she was being watched by her neighbors. Whenever she read in the newspapers of a crime, she said that people in the town thought she was "in back of it." She believed men came into her children's room at night, and thought she had to keep awake to watch out for them. She became over-religious. Self-destruction had been attempted twice. Her return to the hospital was as a voluntary patient. There she continued to express ideas of persecution. When she broke her toe in bowling, she said she had been influenced by the ward physician and that the ward physician was responsible for her injury. In her belief, the examination made at the hospital

caused her to become worse; she thought her husband was talking of getting their marriage annulled and felt this might be of benefit to her. Saying that the doctors in the hospital influenced her will, she complained of mistreatment at intervals during her stay. She would become markedly depressed and, on these occasions, blamed herself for what she called her husband's "political downfall." Expressing suicidal ideas, she said she believed she had been hypnotized, that political parties were persecuting her. At times she assumed a superior attitude toward other patients and employees. Among her charges were that patients had been killed by neglect and that doctors made obscene drawings to influence patients. The food had been tampered with, she said, and all hospital authorities had been antagonistic to her. This was followed by the idea that she was pregnant. Three years after her readmission, she began to show active assaultiveness and resistiveness; became threatening and, at times, destructive. Three years and nine months after her second admission, she contracted lobar pneumonia and died.

Case 4. (*James Roe, Son of Jane and Richard Roe, and Grandson of the Does*) *Third Generation*

Family History: For grandparents and mother, see preceding case histories. The father is engaged in a profession and is in good mental health; two sisters are also in good mental health.

Personal History: The patient is 21 years old and was born in northern New York. He attended boarding school where he was handicapped because of defective hearing, but refused to wear the appliance purchased to aid. Application to his studies was mediocre, and he was resistive to discipline, having the attitude of a boy who had been pampered. He was graduated from high school in June, 1936, and after that time more or less loafed about the house. Unwilling to go out to find work, he attempted to persuade himself that he could qualify for a career by practising art and music, believing that he had more talent than he possessed. The onset of his psychosis, the father said, was about six months before admission. The family first noticed the change in his attitude toward his father and sisters. Whereas previously he had been respectful, he became irritable and stubborn and frequently insulted the rest of the family. Insisting on having his meals at whatever time he

wished, he would eat tremendous amounts of food at night, was stubbornly reluctant to get out of bed in the morning, then would loll about the house all day and play the radio loudly. His family humored him and allowed him to do much as he pleased, but he became more difficult to manage as time went on. He became melancholy, refused to go along with his family to visit relatives at Christmas and, when he ate with the rest of the family at home, acted as if he were starved and displayed marked hatred toward his sisters. At times he refused to talk with members of his family and would make abrupt answers when spoken to.

One week prior to admission, he told his father that the house was too small for him and the father to live in and that he wanted to go away. His father arranged for him to go to another city, and the youth left on January 16. But he unexpectedly returned on January 22. He entered the house rapidly; did not take off his overcoat; sat down, told his father that he was going to California at some future time and that in the interval he was going to stay at his father's house. Asserting that he did not expect any abuse from his father, he told him that, if he attempted to do anything, he would be "pounded to a pulp." There were two reasons, he said for coming home. Revenge was one; the other he would not disclose. Because of this, he was committed to the hospital.

On admission he showed a paranoid trend marked by suspiciousness and hostility directed against members of his family. His father and two sisters had been successfully dominating him ever since he could remember, he said. He was an idealist, he declared, having a feeling for his fellowmen; and, while his father was always lecturing him, the father at the same time was fooling other people and taking advantage of their ignorance. The young man believed that other relatives were making derogatory remarks about him; said that his father ridiculed him; that he was nagged at home continually; that he was dead as far as spirit went; that his family was responsible for his illness. He remains at the hospital at the present time and still entertains paranoid ideas relating to immediate members of his family. For a while he refused to tell his ideas to physicians because he himself said that he would be considered "crazy." Later, he said he had thought since an early age that he had been doped by his father and that, while he

was under the influence of this dope, his father would sexually assault him by rectum. He said his father had continued to do this until he left home and that, judging from the conduct of his sisters, he believed their father had been "carrying on" in a similar manner with them. He said that his father attempted to murder his mother, asserting that once upon his return from school he had found his mother attempting to commit suicide by taking Lysol. His father, he said, was driving toward home simultaneously in a leisurely fashion, and it was his opinion that his father deliberately drove home slowly so that the mother might die before he got there. His father was extremely brilliant, he declared, and the father had influenced the mother to make this suicidal attempt. Also, he said, the father was able to influence the patient through arguing and talking. He declared he had never mentioned this to his father because he was afraid of him. The patient himself has been irritable and threatening. He threatened to commit suicide and has been impulsive and assaultive toward other patients. Physical examination showed a well-developed white male with some impairment of hearing due to an old mastoid operation. A diagnosis was made of dementia præcox, paranoid type.

St. Lawrence State Hospital
Ogdensburg, N. Y.

REFERENCE

1. Meyerson, Abraham: The Inheritance of Mental Diseases.

CHANGES IN THE VASCULAR PATTERN OF THE BRAIN IN EXPERIMENTAL TRAUMA

BY MAX HELFAND, M. D.

In a previous paper[1] on the pathological findings in 22 cases of brain trauma, it was reported that, aside from hemorrhages and softenings, many circumscribed areas of anemic paling were found at a distance from the traumatized zone. These varied in size and intensity ranging from ischemia of single cells to a total necrobiosis of a convolution including ganglion cells and glia cells. In many of these ischemic areas, the blood vessel wall showed no visible structural alteration. Through analogy, with other pathologic conditions, they were ascribed to a physiologic disturbance in the functional performance of the vessel, i. e., to a vasomotor dysfunction.

This conclusion was reached indirectly, since with the usual stains one cannot visualize the fine architecture of the vascular bed. However, the Lephene[2] and Pickworth[3] stain provided a method for the demonstration of the vascular pattern, representing the true condition of the vascular bed. Utilizing this stain, the task was undertaken of ascertaining, experimentally in animals, the occurrence of vasamotor changes in brain following trauma to the skull.

For this purpose, it was deemed advisable to obtain first an average normal vascular pattern of various regions of the brain. Five normal cats were killed by ether anaesthesia and various areas of the brain studied with the benzidine stain. The autopsy material was handled most delicately in order to eliminate the possibility of artificial pressure of the material during manipulation and all stains were performed on fresh material after 48 hours' fixation.

It was found that the best results were obtained with the following technical procedure:

1. Fixation of small tissue blocks in formalin for 48 hours.
2. Brief washing of blocks in tap water and frozen sections cut at 200 microns.

3. Staining for 12-18 minutes in the following solution (solution A) freshly made: 1 gm. of benzidine dissolved in 100 cc. abs. alc. mixed with 0.2 gm. sodium nitro-prusside dissolved in 100 cc. of distilled water.

4. Washing quickly in distilled water.

5. Placing for 15-25 min. in the following solution: (Solution B) 0.2 gm. of sodium nitro-prusside dissolved in 195 cc. of distilled water, to which 100 cc. of abs. alc., 4 cc. glacial acid, and 1 cc. super-oxol, were added.

6. Washing in tap water, dehydrating and mounting in balsam.

Sections from various regions of the normal brain revealed well-stained vascular network in distinctive patterns for the gray and white substance as described by Pfeifer,[4] Pickworth,[3] Eckstein.[5] At no time were there any gross anemic or hyperemic variations. There were, however, a few isolated regions in various parts of the brain which appeared anemic in relation to the rest of the brain. The vascular pattern at the base of the convolution in the bottom of the sulci was typical of such a variation (Fig. 1).

As already pointed out by Campbell, Alexander and Putnam,[6] such areas are considered as physiologic variations most likely due to "relatively lowered metabolic activity in the locality in question." These areas were not found in identical regions in all normal cats examined and did not compare in intensity to the areas of anemia or hyperemia found in pathologic states.

The material used for this experimental investigation consisted, in addition to the normal control, of 11 cats, each of which received trauma to the skull, varying in intensity and number, with different intervals of life between the trauma and the sacrifice of the animal. The intensity of the trauma was determined by a kilo weight dropped on the skull of the animal from a height of three feet. All animals were sacrificed through inhalation of ether and the action of the traumatic agent was always applied to the vertex of the skull, the animal lying on its abdomen.

The following is a tabular summary of the protocols of various experiments.

TABLE 1

No. of the animal	Wt. of the animal	Mode of Injury	No. of injuries	Duration of observation and remarks	Pathological findings
1	2.5	Kilo drop from height of 1½′	1	24 hours. No symptoms	Normal findings in capillary and cellular studies (Nissl stain).
2	2.3	Kilo drop from height of 3′	3	4 days. Cat dazed after the trauma	Skull intact. Capillary congestion in many areas of cortex especially at the border between the gray and white substance. There are many, more or less circumscribed areas within the cortex, wherein the capillary bed is not visualized. This is mostly noted in the supra sylvian gyrus. In the cerebellum there are interruptions in the staining of the capillary network in the granular layer. There are no cellular changes (Nissl stain).
3	2.5	1 kilo drop from height of 3′	21	81 days. Cat dazed most of the time following the trauma. Loss of weight to 1.8	Free blood in subarachnoid space. Congestion of capillary network in frontal and occipital lobes. Disturbance of capillary pattern in gyrus lateralis, showing spotty areas wherein the capillaries are not visualized. Some of the remaining vascular network appears fragmented and disintegrated so that one observes only a granular deposit. These were seen throughout the entire cortex, but most intense in the upper and lower layers (Fig. 2). The Nissl stain shows neuronophagia in the caudate nucleus. There is an organized small hemorrhage involving 1st, 2nd, and 3rd layers of the parietal cortex.
4	2.3	1 kilo drop from the height of 3′	2 consecusive blows in 1 experiment	3 days. Cat is stunned following each experiment	Marked capillary disintegration with granular deposit in many areas especially in parietal and orbital lobes, also in the thalamus and globus pallidus on each side. In the latter, there are many very small hemorrhagic exudations, intermingled with areas of paling. Some of them are circumscribed and disclose in the center, contracted blood vessels. Nissl stain does not reveal pathologic findings.

TABLE 1—(Continued)

No. of the animal	Wt. of the animal	Mode of Injury	No. of injuries	Duration of observation and remarks	Pathological findings
5	2.4	Kilo drop from height of 3′	5 consecutive blows on each of 3 successive days	5 days. Cat stunned following each experiment	Brain congested. Capillary congestion with minute hemorrhagic exudations in many areas especially in parietal lobes (Fig. 3). Capillary pattern in the superficial layers of occipital lobe greatly disturbed showing a reduced number of vessels with many cortical areas in which visualization of blood vessels is completely lacking. Nissl stain shows several diapedetic hemorrhages in white substance of frontal lobe. Ischemic cell changes in Ammon's horn. Circumscribed area of paling in occipital cortex.
6	2.3	1 kilo drop from the height of 3′	5 consecutive blows on each of 2 successive days	2 days. Cat stunned following each experiment	No gross pathology observable. Microscopic vascular pattern considerably disturbed in cortex of supra sylvian gyrus and gyrus orbitalis, which present a general poverty of visualized blood vessels in all layers. Some of the larger vessels are on the contrary plainly outstanding (Fig. 4). Nissl stain shows paling of cells in the cortex of the frontal lobe (Fig. 5) rather diffuse in distribution and involving mostly the upper layers. The character of the cell change was often typical of ischemic changes.
7	2	1 kilo drop from the height of 3′	10 consecutive blows in 1 day	7 days. Cat unconscious for 30 minutes following the experiment	Brain congested. There is localized constriction of capillary bed in confluent areas throughout the entire cortex, more marked in gyrus lateralis. Nissl stain shows circumscribed loss of cells in multiple areas of cortex (Fig. 6). No hemorrhages observable.

TABLE 1—(Concluded)

TABLE 1—(Concluded)

No. of the animal	Wt. of the animal	Mode of Injury	No. of injuries	Duration of observation and remarks	Pathological findings
8	2.2	Cat under anaesthesia, 1 kilo drop from 3′ height	3 consecutive blows in 1 day	1 day. Cat reacted apparently normally	No appreciable vascular or cellular disturbances. Very close to normal.
9	2	Kilo dropped from 3′ height	20 consecutive blows in 1 day	10 days. Cat unconscious 1 hour following the experiment	Many cortical areas of the brain were devoid of capillary network. These areas were more or less circumscribed but at times were confluent, giving the impression of continuity. The larger vessels were slightly visualized. These changes were most severe in ecto-sylvian and occipital gyri. Interruption of capillary pattern in cerebellum with many lobules appearing pale and showing hardly any vessels. No findings in Nissl stain. More often the external layers of the cortex disclosed the most severe damage (Fig. 7).
10	2.4	Kilo dropped from height of 3′	1 blow on every third day for 39 days	40 days. 1.6 weight at death	Many areas showing hyperemia of vascular bed in gyrus orbitalis. Almost complete absence of capillary network in suprasylvian, parietal and occipital gyri. Nissl stain shows circumscribed areas of paling in the occipital cortex (Fig. 8) with fresh softening in white substance. None of the cells in this softening contained evidence of hemorrhagic pigment. Parietal cortex shows evidence of neuronophagia in the deeper layers with ischemic changes of some ganglion cells.
11	2.4	1 kilo dropped from 3′ height	20 consecutive blows in 1 day	4 days. Cat unconscious following trauma	Subarachnoid hemorrhage. Many regions in the entire cortex showing vascular pattern disturbance especially in gyrus lateralis. In these regions there was poverty of visible capillaries with some of the larger vessels embedded in granular deposits. These areas showed a tendency to fuse extending at times to all the layers of the cortex. Marked vascular disturbance in superior layers of the frontal cortex. Nissl stain discloses glia-Strauchwerk in cerebellum. Hemorrhagic necrosis in pons and medulla with local perivascular infiltration.

Comment

From this table it can be seen that, in cats, an adequate trauma to the head may produce a more or less localized disturbance in the regularity of the vascular pattern. This is especially observable in the motor and visual cortex. At times, all the cortical layers of the affected areas are involved, but usually the vascular disturbance is predominant in upper layers of the cortex. The character of the disturbance varies from a complete lack of staining of the intravascular contents to a distortion in the regularity of the caliber of the blood vessels or to a dilatation and engorgement of the vessels. The latter may or may not be accompanied by diapedesis. With the exception of the subarachnoid bleeding, encountered in cats 3 and 11, there is no evidence of large intracerebral hemorrhage. The larger vessels appear to be resistant to functional changes, and are usually detectable even though the capillaries are not outlined.

As already indicated, it is felt that the extreme disturbances in the vascular pattern reported in this study represent pathologic states of vascular performance. These states manifest themselves in spasms of the capillaries resulting in the emptying of the capillaries and the capillary bed, or in dilatation leading to engorgement of the capillaries and surrounding edema. It is felt that these functional states of the vascular apparatus are reversible and that only a prolonged duration of such states might result in permanent damage to the parenchyma. This is evident from the fact revealed in this material that the cellular changes as viewed by the Nissl stain, only occasionally correspond to the vasospasm revealed by the benzidine stain and found only in those cats which sustained the biggest trauma. In animal 3, which received one injury on each of three successive days, there were no pathological findings in the Nissl stain, whereas the benzidine stain revealed various disturbances in the capillary network. On the other hand, in animal 6, which received five consecutive injuries in each of two successive days, there are definite ischemic changes in the cells of the frontal cortex. It seems reasonable to presume that the duration of the functional changes in the blood vessel is directly dependent on the intensity of the injury. Only when the vascular dysfunction lasts for a certain period of time, do the cells suffer in their structure.

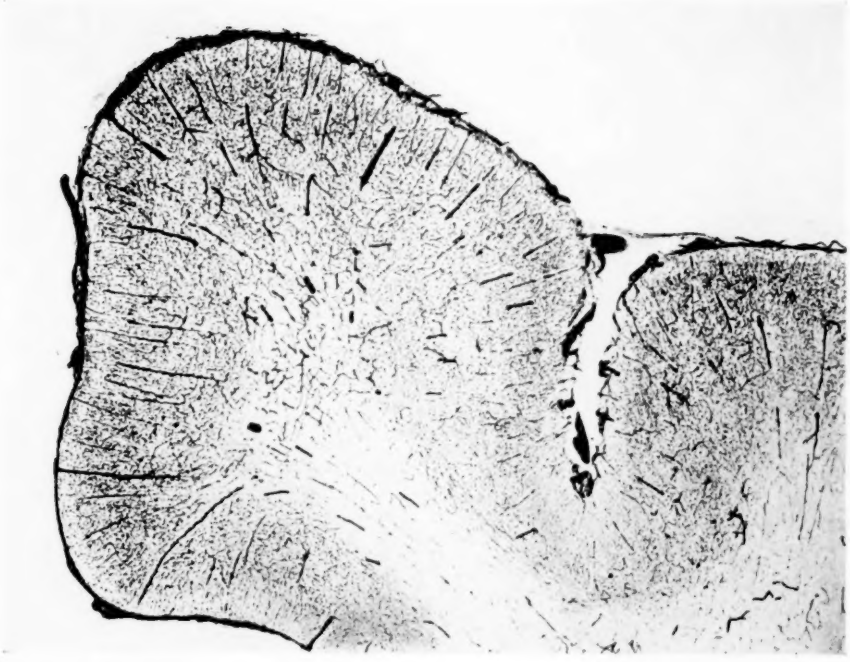


Fig. 1. Benzidine stain showing normal capillary network in gray and white substance. The vessels, both large and small are regularly distributed. The base of the convolution shows a relative poverty of blood supply. This was considered as a normal physiologic variation.

Fig. 2. Benzidine stain showing disturbance of capillary network of Gyrus Lateralis. There are many spotty areas wherein the capillaries are not visualized. Some of the remaining capillaries are fragmented. There is also a local circumscribed engorgement of some capillaries. (Cat No. 3).

Fig. 3. Benzidine stain showing congestion of vascular network with minute hemorrhagic exudations. Greatly disturbed capillary pattern (cat No. 5).

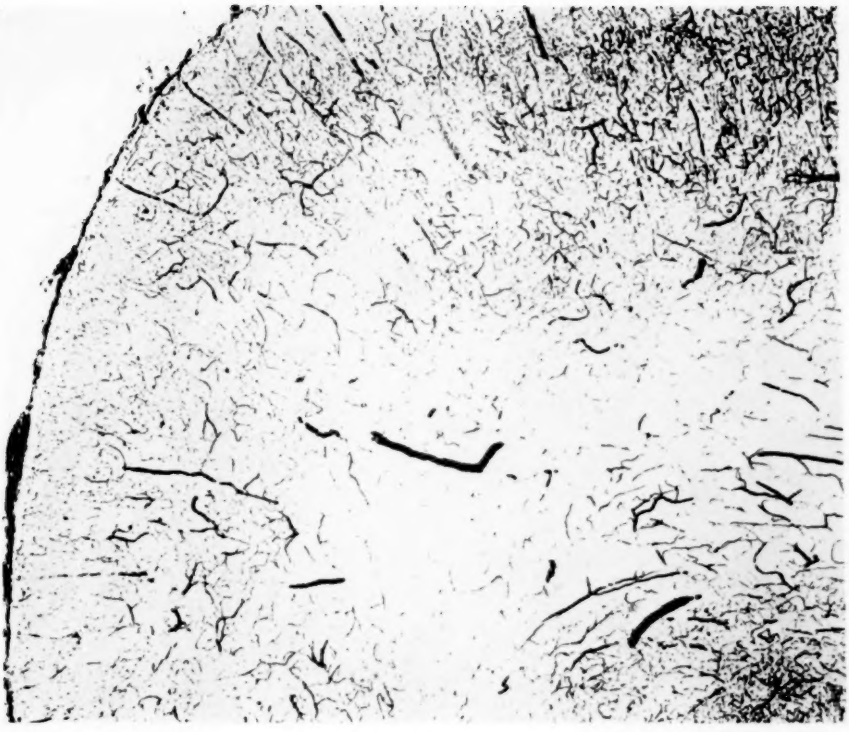


Fig. 4. Benzidine stain showing a marked poverty of visualized capillary network in Suprasylvian Gyrus (cat No. 6).

Fig. 5. Nissl stain showing diffuse ischemic cell changes of the upper layers resulting in paling and cellular rarefaction. Frontal cortex (cat No. 6).

Fig. 6. Nissl stain showing circumscribed area of paling, wherein the rarefied cells exhibit ischemic changes. (Cat No. 7).

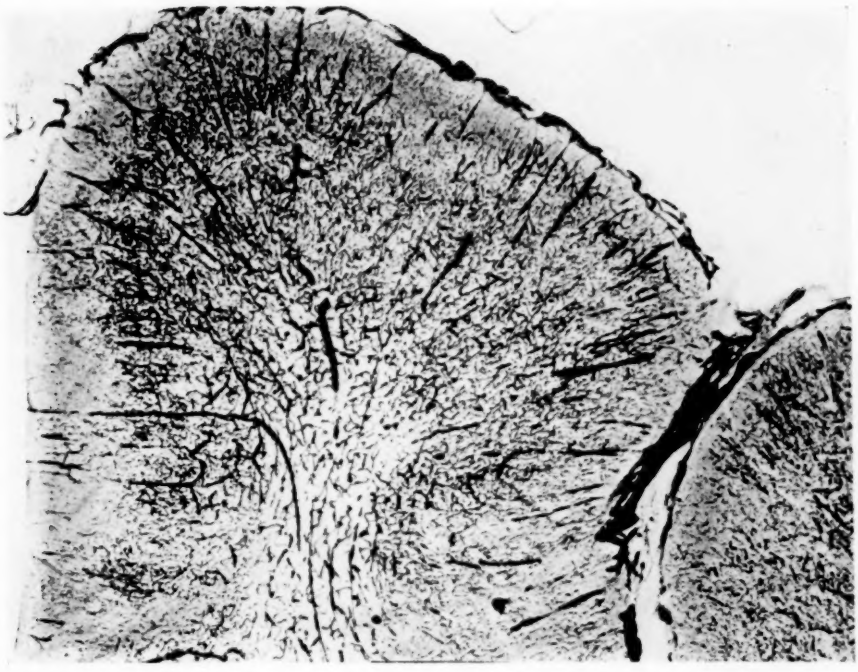


Fig. 7. Benzidine stain. Ectosylvian gyrus showing many circumscribed anemic areas wherein the capillaries are hardly visualized. These areas are more severe and numerous in the upper layers of the cortex. (Cat No. 9).

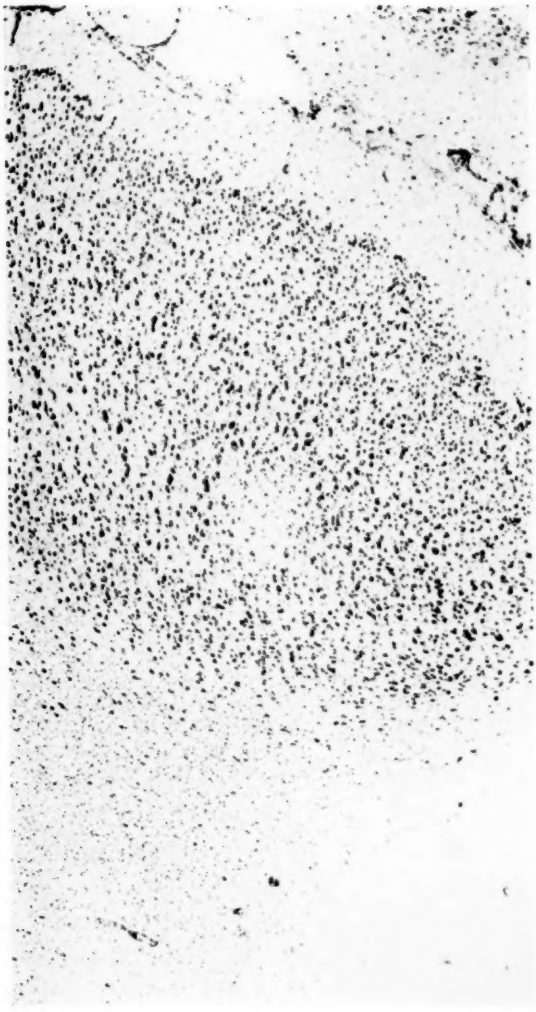


Fig. 8. Nissl stain showing circumscribed area of ischemic cell changes in the occipital lobe of cat No. 10.

In this connection, Weinberger[7] has shown that even in complete experimental obliteration of the blood flow to the brain, no cortical cell damage is demonstrable before two minutes' duration of the obliteration itself. It is conceivable that the spasm produced by the trauma does not compare in intensity and duration with the complete experimental obliteration; and, therefore, the time element essential to cause cell damage must presumably be longer to produce structural changes.

In considering the pathophysiology of these vascular changes, one might attribute them to a dysfunction related to stimulation of a local peripheral vascular component of the autonomic nervous system. That such a mechanism plays an important rôle in the regulation of the cerebral blood flow was contended by Forbes[8] who states that one of the cerebral factors to account for the changes in the caliber of a cerebral artery or arteriole is "the local activity of the neurons." This is made possible by the fact that the cerebral vessels are innervated and influenced by the autonomic nervous system, sympathetic or parasympathetic (Chorbski and Penfield.[9]) The stimulation of parasympathetic fibers in the geniculate ganglia dilate the cerebral vessels, whereas stimulation of the sympathetic fibers in the stellate ganglia constrict the cerebral vessels. Heymans and Bouckaert[10] also attribute to a "local vascular reflex," the dilatation of the cephalic vessels in experiments on isolated head preparations which were supplied with blood from another animal.

It is quite probable that the local vascular reflex disturbances originated by the trauma are transmitted via the meningeal vessels to the superficial layers of the cortex. This might account for the frequent findings of the vascular pattern disturbance in the upper layers of the cerebral hemispheres, especially in view of Campbell's[11] observations that the upper layers of the brain cortex are supplied mainly by short arterioles ending in the cerebral capillary meshwork soon after their entrance from the pia. Campbell also thought this anatomic condition accounted not only for the difference in vascularity but also for the difference in the rate of blood flow between the upper and lower layers of the cerebral cortex in the cat. These observations aid greatly in the explanation of the different traumatic effects on the various cortical layers.

The impact of the trauma may also cause, through vibration, local discrete intracellular changes which might result in structural change or modified metabolic activity. That intracellular changes of a structural nature occur in the course of trauma to the nervous system has been reported by Ferraro[12] who described in his material on experimental trauma, the displacement and expulsion of the nucleolus from the nucleus of the nerve cell. The modified metabolism might lead to increased formation of lactic acid and other by-products. The accumulation of these substances would be absorbed and drained into the cerebral venous system and finally carried to the cerebral arterial system to affect the functional states of arterial walls. However, the writer thinks it more likely that the increased metabolites stimuate locally the nerve endings in the blood vessel walls and influence the caliber of the vessel via a local axon reflex depending on the branching of a peripheral afferent fiber.[13] This explanation tallies with Campbell's[14] findings of a positive correlation between localized functional activity and regional vascularity. Also Scharrer[15] in his work on the opposum brain has shown direct relationship between metabolic activity and capillary density.

Alexander[16] expressed the opinion that the changes in the vascular pattern might be due to a fall in the systolic blood pressure at the time of the trauma. This explanation, strongly applicable to acute functional conditions seems to the writer to be incomplete in explaining the disturbed vascular pattern in animals submitted to repeated blows and sacrificed at various periods of time following the trauma, especially when periods of days and weeks have elapsed between the trauma and sacrifice of the animal.

While a permanent general drop of blood pressure, strong enough to justify the emptiness of the capillary network is difficult to accept, one can more easily conceive of a permanent vascular spasm resulting from repeated traumatic stimuli, causing localized disturbances in certain areas of the cerebrum and cerebellum.

Conclusions

1. Cranial trauma of sufficient intensity produces temporary or permanent diffuse or localized disturbances in the vascular pattern of the brain cortex.

2. These disturbances range from complete local absence of visualization of the capillary network, to local engorgement with diapedetic exudations.

3. These disturbances seem to be the expressions of physiologic dysfunction of the blood vessels and may be reversible, depending on the intensity and duration of the trauma.

4. When the circulatory dysfunction is lasting, the ganglion cells disclose structural changes ranging from ischemic alterations of individual cells, to more or less circumscribed area of global necrosis.

Department of Neuropathology
New York State Psychiatric Institute and Hospital
New York, N. Y.

REFERENCES

1. Helfand, M.: Cerebral lesions due to vasomotor disturbances following brain trauma. J. of Nerv. and Ment. Dis., 90:157, 1939.
2. Lephene, G.: Zerfall der roten Blutkorperchen beim Ikterus Infektiosus. Beitr. z. path. Anat. u. z. allg. Path., 65:163, 1919.
3. Pickworth, F. A.: A new method of study of the brain capillaries and its application to the regional localization of mental disorder. J. Anat., 69:62, 1934.
4. Pfeifer, R. A.: Die Angioarchitektonik der Grosshirnrinde, Berlin. J. Springer, 1928.
5. Eckstein, A.: Untersuchungen über die Angioarchitektonik des Gehirns in fruhen Kindesalten. Ztschr. f. d. ges. Neurol. u. Psychiat., 154:298, 1936.
6. Campbell, A. Colin P., Alexander, L., and Putnam, T. J.: Vascular pattern in various lesions of the human central nervous system. Arch of Neurol. and Psych., 39:1150, 1938.
7. Weinberger, L. M., Gibbons, M. H., and Gibbon, J. H.: Temporary arrest of the circulation to the central nervous system. Arch. of Neurol. and Psych., 43:615, 1940.
8. Forbes, H. S.: Physiologic regulation of the cerebral circulation. Arch. of Neurol. and Psych., 43:804, 1940.
9. Chorobski, J., and Penfield, W.: Cerebral vasodilator nerves and their pathway from medulla oblongata. Arch. Neurol. and Psych., 28:1257, 1932.
10. Heymans, C., and Bouckaert, J. J.: Sur la regulation reflexe de la pression arterielle cephalique. Comp. rend. Soc. de biol., 99:1871, 1928.
11. Campbell, A. C. P.: The vascular architecture of the cat's brain: a study by vital injection. Research Nerv. and Ment. Dis. Proc., 18:69, 1938.
12. Ferraro, A.: Contributo alla conoscenza dell'Anatomia patologica della Distruzione primitiva del midollo spinale. Cervello, Napoli, 1:361, 1922.
13. Krough, A.: Anat. and Physiology of Capillaries. New Haven, Conn. Yale Univ. Press, 1929.
14. Campbell, A. C. P.: Variation in vascularity and oxidose content in different regions of the brain of the cat. Arch. Neur. and Psychiat., 41:223, 1939.
15. Scharrer, E.: The functional significance of the capillary bed in the brain of the opposum. Anat. Rec., 75:319, 1939.
16. Discussion of this paper, "Changes in vascular pattern following experimental trauma of the brain," at the annual meeting of the American Association of Neuropathologists, June 6, 1940, at Rye, N. Y.

AN OBJECTIVE SIGN (GANS-RODIET) IN CHRONIC ALCOHOLISM*

BY M. K. AMDUR, M. D., AND S. T. GINSBERG, M. D.

It is generally conceded that alcohol, taken in large quantities over a long period of time, produces sooner or later serious damage to the individual. It has been claimed that 12 to 15 per cent of the major psychoses are dependent upon alcohol as the principal etiological factor. These figures are questionable, since information concerning alcoholic habits is frequently withheld for diverse reasons. Furthermore, the so-called alcoholic psychoses, one must admit, may depend upon some peculiarity of the individual's makeup. It is not unlikely that in many cases alcoholism is only an expression of these peculiarities. Nevertheless, knowledge of the excessive and habitual use of alcohol is of importance in evaluating the symptomatology in a large number of psychiatric patients.

L. M. Brown[1] has discussed the pitfalls in psychiatric examinations, deploring the scarcity of objective findings in psychiatry. This is of special importance in the examinations of all patients presenting a neuropsychiatric problem, where the fact of the use and abuse of alcohol may be minimized or hidden by the patients, as well as by their neighbors and relatives. Hence, a dependable objective sign of alcoholism would be of great value to neuropsychiatrists.

One objective sign of alcoholism is surprisingly obscure, although its authors described it as far back as 1906. Gans and Rodiet[2] in their studies of alcoholics had found a peculiar hemianesthesia of the external or lateral part of the sclera of the eye; i. e., the lateral part of both eyes loses the sensation of touch, and the motor or defense reflex is weakened or even lost.

In 1908 Obrastzov[3] verified these findings and called special attention to the technique of the test, which requires exactness and constancy in its methods.

Ilin[4] was especially interested in this phenomenon. He ascertained that stimulating the conjunctiva of the sclera provoked a subjective, unpleasant sensation differing in degree under normal

*Published with the permission of the medical director of the Veterans' Administration, Washington, D. C., who assumes no responsibility for the opinions expressed or conclusions drawn.

conditions, as well as a motor reflex in the form of closing the eyelids. Like any other reflex, it varies from exaggeration to diminution and loss.

This reflex arc originates in the nerve endings in the sclera through fibers of the ophthalmic nerve, or first division of the trigeminal nerve, to the trigeminal nucleus, thence to the facial nucleus, and the fibers of the facial nerve to the eyelids.

The diminution of the conjunctival reflex observed in organic as well as in psychogenic disorders of the central nervous system depends upon the level of the involvement of the reflex pathway. In organic conditions, the disturbance of the reflex is usually unilateral; in psychogenic conditions it is usually bilateral; however, in both conditions, the subjective feeling of touch is almost never lost. Clinicians had found that a peripheral involvement of the trigeminal nerve is met with rarely; usually it is unilateral and is accompanied by circulatory disturbances, edema of the conjunctiva, and diminution of its sensitivity. The involvement of the facial nerve which participates in this reflex originates simultaneously with that of the trigeminal nerve, and one of the principal symptoms characterizing neuritis of the facial nerve is the weakening of the conjunctival reflex. In these cases there are also disturbances of hearing, of lacrimation, salivation, etc.

To explain the pathogenesis of the Gans-Rodiet sign, it becomes necessary to understand the distribution of the branches of the ophthalmic nerve, or first the division of the trigeminal nerve. The ophthalmic nerve divides into three terminal branches: the frontal, the lacrimal and the nasociliary nerves. According to Rauber[5] and Morris,[6] the conjunctiva of the sclera and the lid is innervated by branches of the lacrimal nerve and the supraorbital nerve, which is the largest of the branches of the frontal nerve. The terminal branches of the lacrimal nerve which innervate the conjunctiva are limited to the lateral portions of the scleral conjunctiva, while the branches of the supraorbital nerve innervate the medial portion of the scleral conjunctiva.

Thus the lateral and medial parts of the sclera of the eye possess separate innervations. This dual innervation of the medial and lateral halves, according to Ilin,[4] is the basis for the explanation of the Gans-Rodiet sign.

Ilin attempted to study this phenomenon in animals. He used rabbits for his experimentations, and after two to three weeks during which the animals were intoxicated with ethyl alcohol, he had no difficulty in finding lateral hemianesthesia of the sclera of the eyes. If the experiment was made only once and the intoxication was an acute one, the anesthesia of the sclerae of the experimental animal was not prolonged, gradually disappearing in a few hours. Ilin concluded that this was a local effect of alcohol on the conjunctiva of the sclera of the eye in the experimental animal. He directed his attention first to the function of the nearest gland, the lacrimal, whose secretory activity is connected functionally with the trigeminal as well as the facial nerves. The location of this gland, as is well known, is at the external angle of the orbit.

If one recalls that not all the alcohol introduced into the organism is "burned up," but that a certain part of it (up to 10 per cent) is eliminated by the lungs and kidneys in unchanged form, then one has no reason to deny the possibility that in the mechanism of the elimination of alcohol from the body the lacrimal gland participates equally with the other excretory organs. It is known that even dilute solutions of alcohol have astringent and dehydrating action on the protein and epithelia of the mucous and serous membranes. As alcohol is secreted by the lacrimal gland, its effects are limited to the lateral portion of the sclera of the eye. This local toxic effect produces the peculiar hemianesthesia and a resultant loss of the protective mechanism, due to excessive or prolonged use of alcohol.

The technique for the investigation of this sign, as mentioned, is of great importance. True, not only is it difficult to ascertain the loss or presence of the blinking reflex, but one must be exact and constant in his technique to obtain accurate results. The individual being examined must not face a window, since the light from the window, even if it is dim, stimulates a motor response through the retina and frequently produces lacrimation for a short period of time. This, of course, changes the conditions of the investigation as well as the results. It is necessary also to avoid any variation in results which may be due to accommodation. Hence it is important to examine the patient with his back to the window, the examiner standing beside the patient, using a long applicator

and stimulating the eye from the side, so that the patient's eyes are not fixed on the hand of the examiner. However minor these points may appear, they are, nevertheless, of importance in obtaining exact results.

The normal reaction to stimulation of the lateral portion of the sclera of the eyes is a brisk blinking reflex with a tickling, irritating sensation. A positive Gans-Rodiet sign is characterized by diminished sensitivity to total lack of sensitivity and by sluggish blinking to the loss of the blinking reflex.

In Ilin's studies of patients whose anamneses indicated alcoholic intoxications, he found the Gans-Rodiet sign in 72 to 80 per cent of the cases.

The writers carried out the exact technique emphasized by Obrastzov in studies of the Gans-Rodiet sign on 300 patients, 100 of whom had a definite alcoholic history, and 200 whose anamneses indicated very light or no alcoholic indulgence. Sixty-two of the 100 patients whose anamneses indicated alcoholic intoxication had a positive Gans-Rodiet sign. These cases showed various degrees from moderately diminished sensitivity to a total lack of sensitivity, and from diminution to loss of the protective blinking reflex of the eye. This sign was found to be bilateral in all 62 patients.

Only 15 (or 7½ per cent) of the 200 patients without history of alcoholism revealed anesthesia of the sclera or inhibition of the protective mechanism. Of these 15 cases, 12 had anesthesia of the entire sclera, both medial and lateral portions. On further study of these 12 cases, all were found to have syphilis of the central nervous system. It is of interest that of the 300 cases examined, only two had definite hypersensitivity of the lateral portion of the sclera, with a vigorous protective reflex; both of these were cases of acute alcoholism combined with neurosyphilis.

The writers believe that the Gans-Rodiet sign is an important objective test for chronic or even acute alcoholism, and that it could be of great value to neuropsychiatrists in the objective knowledge of prolonged use of alcohol by patients under examination. It would seem advisable that this test be performed and recorded in every neuropsychiatric examination.

Conclusions

1. The history and technique of the Gans-Rodiet sign is given.

2. A plausible explanation of the mechanism of this sign has been advanced.

3. Three hundred cases (100 with anamneses positive for alcoholic intoxication, and 200 with no history of alcoholism) were examined for this sign, and findings recorded.

4. The Gans-Rodiet sign is considered an important objective test for chronic and even acute alcoholism, and is of value to neuropsychiatrists.

Veterans' Administration Facility
Coatesville, Pa.
Augusta, Ga.

REFERENCES

1. Brown, L. M.: Pitfalls in psychiatric examination. U. S. Vet. Bur. Med. Bull., January, 1936.
2. Rodiet, A., and Gans, F.: Diagnostic différentiel des troubles cérébraux d'origine toxique dus à l'alcool et au tabac et de la paralysie générale d'après les symptômes oculaires. Arch. Méd. Psychol., IV:408-420, 1906.
3. Obrastzov, V. N.: The symptomatology of alcoholism. "Sovrem. Psikhiat." Mosk., 1908, v. 2, 7-15 (in Russian).
4. Ilin, A. B.: On the problem of differential diagnosis in alcoholic intoxication. Jour. Neuropathol. and Psychiat., 1, 1931, Moscow-Leningrad (in Russian).
5. Rauber's Lehrbuch der Anatomie des Menschen (herausg. von F. Kopsch). 11 Aufl., 1920, Abt. 6.
6. Jackson, C. M.: Morris' Human Anatomy. 9th ed., 1933.

STUDIES IN SENILE NOCTURNAL DELIRIUM*

BY D. EWEN CAMERON, M. D.

Nocturnal delirium is a phenomenon which is frequently encountered in senile patients. In many instances its appearance constitutes the final reason for admittance to hospital. The following facts are already known concerning it.

The delirium usually appears after retiring to sleep and clears up soon after getting up the next day. During the delirium there is a complete disorientation; frequently some degree of agitation and panic is apparent; and in the more severe forms destructiveness and incontinence appear. Information concerning the reaction is limited, and no reliable data concerning the possible etiology are available (Bleuler, 1936; Sadler, 1936; Henderson and Gillespie, 1937; Noyes, 1939).

Preliminary to setting up an experimental investigation of this phenomenon, it was necessary to determine whether the onset of darkness was a major causative factor or whether one should consider an accumulation of fatigue during the day as an etiological possibility. To check this each patient was put in a dark room during the earlier part of the day, long before his delirium customarily appeared. In every instance delirium appeared within an hour after the patient had been put in the dark room, and in some cases a degree of agitation also became apparent. This subsided again in about an hour after the patient was brought back into the light. The "dark room" delirium appeared earlier and was more marked in those patients in whom it was most severe during the night hours. This clearly emphasized the importance of darkness, rather than fatigue, as a causative factor.

Further investigation showed that these patients suffered from marked impairment of recent memory. This was so severe that reproduction of an event could not be correctly carried out within 15 minutes after observation of it. This resulted in a considerable impairment of the patients' capacity to orient themselves. They could not retain the identity of members of the staff unless they saw them constantly. It was easier to retain the identity of the nurse during her period of duty on the ward than that of a physician who appeared less frequently. The patients could not locate

*From the department of neurology and psychiatry, Albany Medical College, Albany, N. Y.

themselves in time, although information on this point was given frequently. They could, however, to some extent locate themselves in the immediate surrounding space, in that, if questioned at once, they could with their eyes closed tell where various objects on the ward were located.

It was conjectured that, since a severe memory defect had resulted in such marked impairment in orientation for time, and in large degree for persons, relative preservation of orientation for the immediate surrounding space might be due to the fact that the spatial image was constantly restored by the view of surrounding objects. In brief, it was postulated that the preservation of a spatial image might in these patients be very much more dependent upon vision than is the case in a normal individual who, after once seeing his surroundings, is able to retain an image of them and of their relationship to him for a considerable period without having constantly to refresh it by looking around him. To test this postulate the following experimental situation was set up.

Procedure

The patient was seated in his own room and asked to point out the location of five common objects in the room—a stand, door, mirror, window, bed—this was done first with the eyes open. Following this, he was blindfolded and was then asked every 15 minutes for an hour to point to where he thought the various objects were located. His answers were recorded. This procedure was carried out on 16 patients suffering from various degrees of nocturnal delirium. In 10 of them, pulse and blood pressure records were taken before and at the end of the hour's period of blindfolding. This was done with the anticipation that, should the agitation which is frequently apparent during nocturnal delirium appear during the period of blindfolding, it would be reflected in the blood pressure and pulse.

Results

In all but three of the 16 patients definite displacement of the objects occurred. Moreover, the displacement occurred earlier and was more marked in those patients who showed the more severe nocturnal delirium. It was noted also that displacement of the objects usually took a forward direction, so that things which were actually located behind the patient or at his sides were finally said

by him to be in front of him. This gradual progression forward of the objects in the patient's space image did not show definite laterality, i. e., objects placed in his right rear did not gradually move up the right side toward the front in his image, but might be first located to the left and then to the front and right before finally appearing in the front of the image. (Figs. 1 and 1-a.)

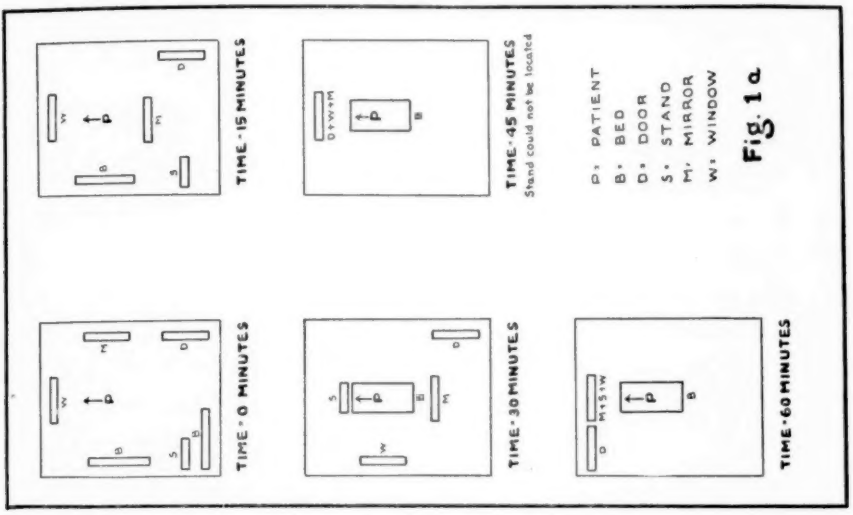


Fig. 1a

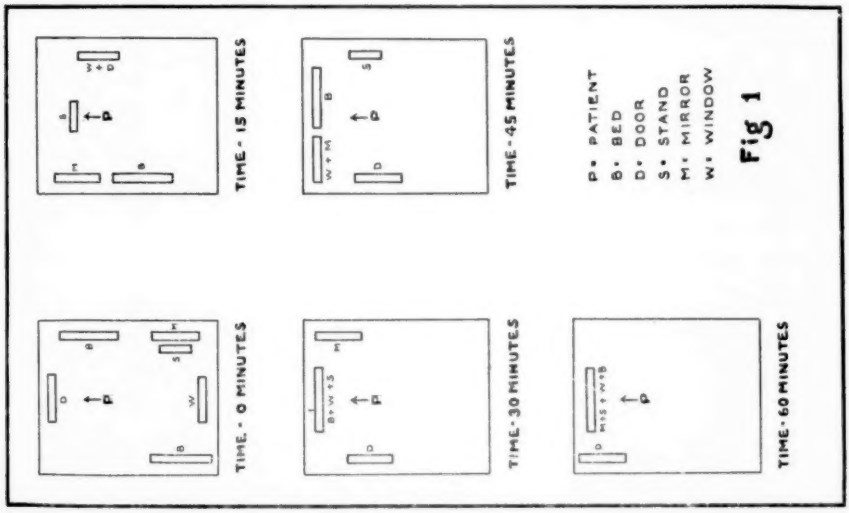
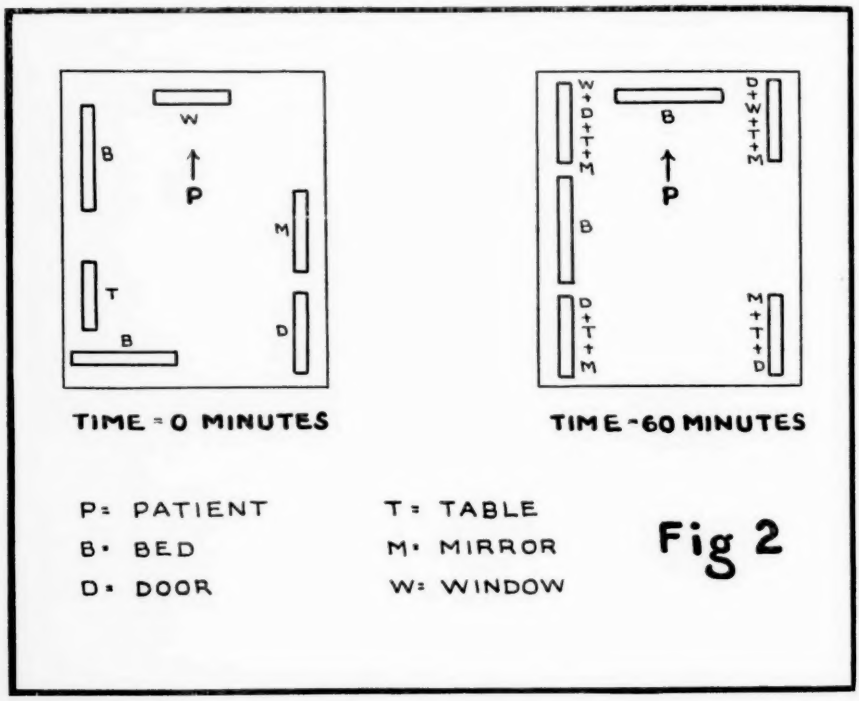


Fig 1

An experiment on one patient, using only one object, also failed to show that the object tended to adhere to one side in its passage to the front of the patient's image.

It was also found that a majority of those who showed disturbance of the location of objects also reported multiplicity: Instead of one bed, two or three would be reported; on one occasion as many as eight windows were stated by the blindfolded subject at the end of an hour to be present in the room, in place of the one which was actually present. (Fig. 2.)



Of the 10 patients in whom pulse and blood pressure rates were recorded, eight showed a rise in blood pressure, this being particularly marked with respect to the systolic blood pressure. The pulse did not vary. One patient showed no change, and in one the blood pressure fell. Average initial blood pressures and pulse rates for the eight showing a rise were 135/85 and 91. Averages after they had been blindfolded for an hour were 151/87 and 91.

Finally, it is to be reported that, after the blindfolding was removed and the patients saw to what extent distortion of their space image had occurred, there was very little distress—acceptance of the fact seemed strikingly facile.

Discussion

From the data obtained it appears that in the patients who were examined maintenance of the spatial image was greatly impaired and failed whenever that maintenance became entirely dependent upon memory. So long as the spatial image could be refreshed by direct visualization, it was possible to preserve it fairly satisfactorily.

It is suggested, therefore, that these experiments sustained the postulate originally put forward, namely, that nocturnal delirium of the senile patient is primarily due to the fact that the severe retention defect, and more particularly the greatly accelerated secondary elaboration (Cameron, 1940) found in senile patients, does not permit of the preservation of the spatial image after darkness has interfered with direct visualization. It seems reasonable to conjecture that, where the patient is no longer able to locate himself in space, he begins to experience insecurity, and a feeling of anxiety develops. This is perhaps borne out by the rise in blood pressure which was found. It is a matter of common observation that emotional disturbance in senile individuals tends to interfere even more than in normals with adequate thinking. Consequently, one may suggest that the emotional disturbance produced by the loss of a spatial image tends still further to increase the confusion.

With regard to the tendency of objects to move forward in the spatial image, the following hypothesis is suggested. On attempting to localize an object with the eyes blindfolded, the patient turns his head and eyes slightly in the direction in which he believes the object to lie. This movement is not complete, i. e., he does not turn completely towards an object which is behind and to the right, but only partially to the right. At the next questioning his recollection of seeing the object is 30 minutes old, while that of turning to the right is only 15 minutes old. Consequently, this

latter recollection is more likely to be reproduced. The head and eyes are again turned in the direction which they took on the last occasion, but again stopped a little short of the point where they stopped on the first occasion. In this way, recollection displaces the object steadily forward in the spatial image. Against this hypothesis stands the fact that it was not possible to demonstrate that the object moves forward consistently on the same side.

The question of multiplicity of images of the same object may conceivably be due to breakdown of the original memory synthesis. It was pointed out in the work already quoted that one of the functions of memory is to produce from multiple registrations a single image which in actuality represents a synthesis of several registrations. The patients on entering a room see the objects from several different angles, i. e., register several different images. These are at the outset synthesized into a single image of each object. It is suggested that in senile patients this ability to maintain a synthesis may be impaired and that in consequence the synthesis rapidly breaks down into its component parts. If this is the case, it would constitute an additional factor in the rapid deterioration of recollection in the senile.

A second explanation which appeared to be valid in a few of the cases was that the patient was unable to keep his spatial image of the particular room separate from his images of rooms which he had registered much more frequently and over a long period of time, e. g., his own room at home. Contamination of the image took place, and under such circumstances the blindfolded patient would describe doors opening out of the room into other parts of what was clearly his own home.

Summary

1. Senile patients suffering from nocturnal delirium were found to develop this delirium on being placed in a dark room during the day.

2. They were also found to suffer from severe impairment of their capacity to retain what they had registered.

3. It was suggested that their delirium may be based upon an inability to maintain a spatial image without the assistance of a repeated visualization.

4. Thirteen out of 16 of these patients when blindfolded showed a severe distortion of their spatial image within an hour. This supports the above hypothesis.

Albany Hospital
Albany, N. Y.

REFERENCES

Bleuler E.: Textbook of Psychiatry. 1930, Macmillan, New York.

Bleuler, E.: Textbook of Psychiatry. 1936, Macmillan, New York, p. 279.

Cameron, D. E.: Certain aspects of defects of recent memory occurring in the psychoses of the senium. 1940, Arch. Neur. & Psychiat., 43, pp. 987-992.

Henderson, D. K., and Gillespie, R. D.: Textbook of Psychiatry. 1937, Oxford Med. Publications, fourth edition, p. 344.

Noyes, A. P.: Modern Clinical Psychiatry. 1939, W. N. Saunders Co., Philadelphia and London. p. 294.

Sadler, W. S.: Theory and Practice of Psychiatry. 1936, C. V. Mosby Co., p. 910.

PROGNOSIS OF HEBEPHRENIA*

A Study of Onset and Clinical Manifestations

BY BERNHARDT S. GOTTLIEB, M. D., MED. SC. D.

In investigating 100 cases of hebephrenic schizophrenia at the New York State Psychiatric Institute, attention was directed to symptomology, clinical course and clinical manifestations in search of factors which might be utilized advantageously toward the determination of prognosis. The cases selected had already been diagnosed by the staff of the Institute as "unmistakable" hebephrenia, in keeping with the standards of the American Psychiatric Association. Only those were considered who were native urban adults of high school education, free of physical defects. Only first admissions were included. Each patient was a resident of the Institute from 1929 to 1933, and each was followed for a period of five or more years.

The investigation disclosed that numerous symptoms made their appearance with regularity for varying periods prior to the usually accepted time of onset of the disorder. Inasmuch as the study was primarily one of prognosis, it became essential that all symptoms and the variations of the clinical course be considered with this in view.

It would be exceedingly useful if one could predict the prognosis of hebephrenic schizophrenia from the clinical symptomatology in the acute phase of the psychosis. In the monograph of Hoffman,[1] reference is made to the investigations of numerous authors (Kraepelin, Stransky, Meyer) who have attempted to determine a single specific symptom in schizophrenia as of prognostic value. Mauz[2] alone has apparently met with some measure of prognostic success, the clinical registration of the actual symptoms.

The schizophrenic patient is recognized by changes occurring in his emotional manifestations, in his activities, or in his intellectual performance.[3] Often these are accompanied by carelessness with reference to the person. Further change may be evident in the

*This article is a report on part of an original investigation, made at the New York State Psychiatric Institute and Hospital, in partial fulfillment of the Doctor of Medical Science Degree at Columbia University.

presence of depression, restlessness and irritability which may result from the patient's realization of his inefficiency. Mental symptoms, although manifestations of specific safety devices adopted by the patient, have little significance apart from the setting in which they have developed. The ideas expressed are indicative of disorders in thought content and in thinking. The patient as a rule is not entirely aware of any defect in his thought, for much of it is controlled by the unconscious; and the repressed material constitutes an important part in the formation of the thought content.

The usual account of hebephrenic schizophrenia refers prominently to the tendency to silliness, smiling and laughter which appear inconsistent with the ideas expressed; peculiar neologisms, hallucinations, delusions, disintegration and deterioration. This picture is just, but gives no insight into the sequence in which symptoms occur, nor does it make possible the recognition of the incipient hebephrenic schizophrenia. With this in mind, the "evolution" of a hebephrenic is presented.

The disorder presents a prodromal period, an acute stage, a stationary stage, and a chronic stage.

Prodromal stage: The disorder usually begins insidiously. The patient at first manifests a feeling of lassitude, weakness, fatigue, loss of appetite and insomnia. There are numerous indefinite physical complaints. Headaches, dizziness, palpitation and fainting may occur. The individual becomes aware that something is wrong with him. He becomes depressed, sad, downhearted, unhappy, anxious, irritable, annoyed, agitated and apprehensive. Although he is likely to have had a schizoid personality for a number of years, he becomes even more shut-in, usually being concerned largely with himself. He associates less with family and friends. There is constant examination of the features, an outstanding symptom being gazing in the mirror to detect changes in facial and body features. The patient is unproductive, suspicious, somewhat absentminded, and is forgetful or negligent of his duties. In conjunction with the agitation and apprehension, he develops twitches of the face and body. There is evidence of grimacing. Toward the end of the prodromal stage, the patient becomes more seclusive, loses interest in his environment, is withdrawn from reality, and becomes deeply preoccupied with various ideas and thoughts. His

suspiciousness has progressed to a feeling that people are looking at him, talking about him, making fun of him, or in some way influencing him. The patient is neat, clean and orderly. During this period he is well oriented as to time, place and person, the sensorium remains clear, but there is evidence of impaired judgment. During this period he is apt to indulge in piano playing or singing at 2 a. m. without consideration for others. He presents few ideas, and stubbornly rationalizes his acts or thoughts. With his preoccupation, he may be observed laughing to himself or weeping without apparent cause. He is now sufficiently disordered to enter the acute stage abruptly. This is the typical form of the prodromal period, which may last from a few months to a few years without advancing to the acute stage.

There is also an atypical prodromal stage. This differs from the typical onset in that it is of shorter duration, hence a rapid advance of symptoms. The patient may present a depressed, suspicious and irritable makeup. He may become seclusive, withdrawn, preoccupied, lose interest in his environment and show evidence of hypochondriasis, but he usually becomes markedly confused and disoriented as to time and person, and he shows inability to concentrate. He appears untidy, unkempt, laughs and cries without apparent cause. He shows a tendency to become stubborn, difficult, insubordinate, unrestrained, restless and loquacious.

Acute stage: The typical hebephrenic schizophrenia is characterized by an accentuation of seclusiveness, withdrawal from reality, loss of interest in the environment. But during this stage many patients develop numerous misinterpretations. Events or activities in the environment, which in fact bear no relationship to the patient, possess some meaning to him. He believes he is suffering invidious discussion. He may feel slighted, annoyed or definitely wronged. Any undesirable aspect, tendency or quality which he will not consciously own may be projected into a delusion of persecution. These ideas are derived from rejected trends and are invested with hatred and bitterness. Everything is expressed as different from its actual condition. People and things do not exist as such but rather in a state acceptable to the ideas and thoughts of the psychotic. The patient may have the delusion that a part of his body has been destroyed or that people are dead. He

presents a feeling of subordination and control by some person or abstract force in the environment. He rationalizes undesirable sex acts committed either by others (in order to punish them) or by himself, or as a wish-fulfillment. A male patient may think he has become impotent through self-pollution, and that this can be detected in his face; the female patient has an idea that someone lies on her every night, gentlemen being sent to her for this purpose. A perplexed, confused state may be present, with ideas of futility, guilt and self-accusation. Or there may be religious trends, or ideas of unworthiness with a subjective sense of loss of social value. The hypochondriasis becomes more marked, and the patient develops somatic delusions: He no longer possesses a brain, or the brain is shrinking; something has happened to his head, he can feel his head getting smaller; the body has died; he no longer exists; only the left side of his body has died; he can tell that the left side of his body is changing its features into the features of the opposite sex; the body is splitting in half, right through the middle. These delusions are accompanied by hallucinations. Auditory hallucinations are most common, but there may also be visual hallucinations, especially in connection with religious trends. Olfactory hallucinations accompany ideas of guilt and sin: The typical "foul odor" of the seminal discharge is constantly present, and not only is the patient aware of it but he is certain that everyone else is aware of the undesirable odor. There is much overt masturbation. The patient is likely to be sexually excited, to expose himself and to be assaultive. Homosexual panic often develops. The affect is disturbed, resulting in discrepancy between emotions and thought content. The patient indulges in much autistic thinking. Nevertheless, he remains completely oriented as to time, place and person. The sensorium is clear, but the patient is lacking in good judgment and has little or no insight into the situation in which he finds himself. He is usually neat and orderly in appearance, and may have a tendency to present immature attitudes: He will become childishly hilarious, peevish, impertinent, rough and rude. Mood and conduct may vary even from day to day, depending upon the extent of the delusions or the "activity" of the hallucinations.

The patient may be docile and childlike, then suddenly become repellent, resistive and irritable. He may be now verbose and loquacious, now taciturn and mute.

The acute stage of the atypical onset is usually characterized by much confusion during the first few days only, but soon the patient again becomes oriented and the disorder follows a course similar to that of the typical form.

Stationary stage: There is nothing unusual in this stage, except that the patient continues to show the symptoms of the acute stage, with periods varying from a few days to a few weeks in which he is improved. Delusions and hallucinations recede to the degree that they are not troublesome. The patient appears more interested in those about him. The staring, preoccupied attitude is less in evidence. The patient appears to be getting well, only to have a sudden reversal with a reappearance of the symptoms, perhaps with even greater confusion and disorientation than previously. These alternating periods may continue for months or years.

Chronic stage: This stage is characterized by rapid disintegration and disorganization of the personality. The patient rapidly withdraws from reality and develops an autistic existence. Reality does not exist, not even the reality of his own person (depersonalization). He may refer to himself in the third person or repeat his own name in full without awareness of its identity to him. The affect may be completely separated from the thoughts: For example, one patient who was ordered to perform certain duties carried them out while screaming constantly, but retained a blank facial expression. There are also numerous evidences of regression to early childhood, such as shameless wetting and soiling. Inanimate objects become animate: "The tree is talking to me," etc. There is childish thinking. Experience and learning processes no longer exist. There is no drive or urge. The patient is given to loquaciousness, usually characterized by a jumble of words and thoughts, a veritable word salad; or he coins new words by condensing syllables of two or more words. He may repeat continually certain phrases or sounds. The earlier delusions or hallucinations usually recede and no longer constitute part of the conflict. Even the early ideas no longer are factors in the disorder. The patient usually has a voracious appetite or is highly selective. He forgets the

functions of the table utensils from one meal to the next and must be shown how to eat. He is likely to be extremely unmannerly, eating rather in primitive animal fashion. During this stage, it is common to find the patient making faces, assuming peculiar attitudes or making strange gestures. His writing presents disconnected trains of thought, shallowness of content and slovenly external form. Much repetition occurs in certain letters or sounds; eradications and alterations are numerous. The patient's silly conduct may become more pronounced, or he may become dull and appear entirely devoid of thought. Progressive deterioration takes place.

The prognostic significance of sudden or insidious onset: It has already been stated that the time of onset of the psychosis is usually set at the moment when the patient or members of the family first became aware of some change in the behavior or personality of the patient. Under the clinical syndrome, hebephrenic schizophrenia is described as having a typical insidious onset or an atypical, stormy onset with the patient showing some confusion. The 100 cases studied here were examined to determine the respective number of typical and atypical onsets and the recovery rates. The total number of cases having an atypical prodromal stage was 13. Of this number, three individuals recovered, four showed improvement, and six remained unimproved. (See Table 1.)

TABLE 1. COMPARISON OF CASES OF ACUTE AND INSIDIOUS ONSET

	Acute onset	Insidious onset
Total number of cases	13	87
Recovered	3	5
Improved	4	13
Unimproved	6	69

It is apparent that the cases of acute onset show a greater recovery rate. This conforms to the findings of Langfeldt,[4] Mauz,[2] and Strecker and Willey.[5] Hence it may be stated as a prognostic indicator that "An acute, atypical, prodromal stage of hebephrenic schizophrenia is conducive to greater recovery or improvement."

The difficulty in making an abstract of symptoms from the records of 100 cases is apparent. The varying ability of internes to recognize and record the presence of certain symptoms and the tendency of some psychiatrists to accentuate particular symptoms

as sufficient for making a diagnosis, without recording "all the symptoms," make the final tabulation less accurate. Nevertheless, all records were carefully examined in the hope of uncovering symptoms in the daily observations, as well as in the mental examination and anamnesis. These are recorded below in Tables 2, 3 and 4.

TABLE 2. CHARACTER OF IDEAS EXPRESSED AND NUMBER OF TIMES RECORDED (ON THE BASIS OF 100 CASES)

Ideas of reference	85	Hallucinations:	
Ideas of persecution	9	Auditory	56
Ideas of influence	17	Visual	19
Ideas of unreality	11	Olfactory	5
Ideas of sex	13	Gustatory	3
Ideas of futility	9	Delusions	62
Hypochondriasis	54	Religious trends	11
Feelings of guilt	8	Ideas of grandeur	2
		Autism	9
		Poverty of ideas	4

TABLE 3. MOOD CHANGES AND NUMBER OF TIMES RECORDED (100 CASES)

Depressed	53	Apathetic and indifferent	9
Apprehensive	28	Anxious	10
Irritable	29	Agitated	16
Perplexed	27		

TABLE 4. NUMBER OF TIMES COMMON SYMPTOMS OF HEBEPHRENIA APPEARED (AT TIME OF ADMISSION)

Withdrawal from reality	89	Lack of concentration	70
Preoccupation	91	Silly giggling	47
Seclusiveness	88	Grimacing	40
Loss of interest	75	Mirror-gazing	49

The above tables are self-explanatory. It is evident that many of the patients expressed multiple ideas. The figure for each symptom is not to be interpreted as necessarily indicating that the symptom did not exist in the remaining cases, but rather that it was never observed and recorded there.

The mood changes as tabulated occurred not only during the acute stage but more especially in the prodromal stage. It is on the basis of all the symptoms mentioned, together with a knowledge of the order of their appearance, that the clinical syndrome of hebephrenic schizophrenia was presented.

In an attempt to evaluate the prognostic significance of symptoms presented by the hebephrenic, all cases were studied with this in view; and the following results were obtained:

Prognostic significance of the symptomatology: Tables 5, 6 and 7 are self-explanatory.

TABLE 5. CHARACTER OF IDEAS EXPRESSED

	Recovered	Improved	Unimproved
Ideas of reference	8	13	64
Ideas of persecution	0	1	8
Ideas of influence	0	3	14
Ideas of unreality	2	1	8
Ideas of sex	1	2	10
Hypochondriasis	4	9	40
Somatic delusions	8	9	45
Auditory hallucinations	5	8	43
Visual hallucinations	1	4	14
Olfactory hallucinations	1	1	3
Gustatory hallucinations	0	0	3
Religious trends	0	2	9
Ideas of futility	1	0	8
Delusions of grandeur	0	0	2
Feelings of guilt	1	2	5
Poverty of ideas	0	0	4

TABLE 6. MOOD CHANGES

	Recovered	Improved	Unimproved
Depressed	5	13	35
Apprehensive	3	8	17
Irritable	1	4	24
Perplexed	2	4	21
Apathetic and indifferent	0	0	9
Anxious	2	4	9
Agitated	2	4	10

TABLE 7. COMMON SYMPTOMS OF HEBEPHRENIA

	Recovered	Improved	Unimproved
Withdrawal from reality	7	19	63
Preoccupation	6	19	66
Seclusiveness	5	16	67
Loss of interest	8	15	52
Lack of concentration	7	11	52
Silly giggling	5	7	35
Grimacing	5	7	28
Mirror-gazing	3	4	12

Of those who showed an inappropriate or inadequate affect in relation to thought content, seven recovered, 13 improved and 48 remained unimproved.

It is apparent from the tables that no one symptom can be considered as of either favorable or unfavorable prognostic significance.[6] A number of authors have attempted to evaluate symptoms in the light of prognosis with similar results. As stated, the symptoms *per se* indicate little, but the manner of their appearance is of considerable importance. The atypical, acute, confusional, depressed state, a fundamental schizophrenic state, favors a good prognosis.

In 1910, Hoch described the "shut-in personality" as occurring in 51 to 66 per cent of his cases of dementia præcox.[7] In his early description, he says: "These persons who do not have a natural tendency to be open and to get in contact with the environment, who are reticent, seclusive, who cannot adapt themselves to situations, who are hard to influence, are often sensitive and stubborn, but this, more in a passive than active way, who show little interest in what goes on, often do not participate in the pleasures, cares and pursuits of those about them; although often sensitive, they do not unburden their hearts, are shy and have a tendency to live in a world of fancies."

Kraepelin,[8] however, does not accept these personality traits as mere elements of prepsychotic temperament. He believes they represent the onset of the disease itself, even though they may make an appearance in earliest childhood. In keeping with the ideas presented by Kraepelin, the writer would conclude from his investigation that the early manifestations of hebephrenia should be viewed in the light of a prodromal stage of the disease rather than as prepsychotic factors.

Summary and Conclusion

One hundred cases of "unmistakable" hebephrenic schizophrenia admitted to the New York State Psychiatric Institute during the years 1929-1933 were analyzed from the standpoint of potential prognostic indications.

These cases were considered from the point of view of mode of onset, symptomatology and clinical course. As a result of the investigation, a more complete clinical syndrome is presented. This consists of a prodromal period which may be of insidious or of acute nature, followed by an acute stage of the disease, a stationary stage and a chronic stage. Recovery or improvement may follow the acute or stationary stages.

In studying the cases from the point of view of nature of onset, it was shown that cases of acute onset show a greater recovery rate. Cases of insidious onset may be considered as having a poor prognosis. The symptomatology of the cases proved of no significance for prognosis. One symptom, however, that of "mirror-gazing" appeared frequently and as one of the earliest manifestations of the disorder. Insufficient attention has been paid to this symptom.

Early recognition of faulty habits and training in childhood, with easy accessibility to mental hygiene clinics so that prompt adjustments can be made, will tend to reduce the number of individuals developing hebephrenia.

225 W. 86th Street
New York City

REFERENCES

1. Hoffman, E.: Prognose und Heilung der Dementia Precox. Monographie Grosshirn in Sachsen, 1919.
2. Mauz, F.: Die Prognostik der Endogen Psychosen, 1-40, Leipzig, 1930.
3. Henderson and Gillespie: Textbook of Psychiatry, 86, Oxford Press, 1936.
4. Langfeldt, G: The Prognosis in Schizophrenia. Monograph, 45, Levin and Munksgaard, Copenhagen, 1937.
5. Strecker and Willey: Recoverable dementia præcox reactions. Am. Jour. Psychiat., 1924, 3, 593.
6. Sullivan, H. S.: Criteria of malignancy in dementia præcox. Am. Jour. Psychiat., 1928, 7, 759-787.
7. Rowe, C.: Comparative study of personality traits in early and late dementia præcox. State Hospital Quarterly, 1921, 6, 167.
8. Kraepelin, E.: Dementia præcox and paraphrenia, 94, Livingston and Co., 1919.

ALCOHOLISM AND HALLUCINOSIS

BY SAMUEL C. KARLAN, M. D.

The relationship of alcoholism to hallucinosis has been the subject of frequent discussion. Some have felt that alcoholic hallucinosis was a mental symptom which was specifically caused by the toxic effect of alcohol. On the contrary, others gained the impression that the hallucinations had little to do with the toxic effect of alcohol but were for the most part coincidental in patients who were constitutionally predisposed to both alcohol and hallucinosis. Henderson and Gillespie[1] stated, "Alcoholic hallucinosis and alcoholic paranoia are probably misnomers, alcohol being a symptom of an underlying instability which gives rise to the syndrome rather than a cause of the latter. We have certainly known precisely similar hallucinoses to occur in the absence of alcoholism." In fact, they noted that at times the hallucinosis was caused by a mental factor solely and at times by the withdrawal of alcohol. Schneider[2] stated that alcoholic hallucinosis seemed to be closely allied to manic-depressive psychosis. Burns[3] noted that in the same patient, alcohol produced at one time an alcoholic psychosis, especially hallucinosis, and at other times typical manic attacks. It might even produce a psychosis which at the outset resembled an alcoholic psychosis and ended as a manic attack. Kirby[4] examined 102 cases and concluded that hallucinosis belonged to the category of reaction types dependent on constitution and emotional situations in the presence of alcoholic excesses. He believed that emotional factors were of great importance as precipitating causes.

The type of personality in which both alcoholism and hallucinosis are prone to occur has been well described by Davidson.[5] It is characterized by a lack of elasticity of affectivity which interferes with adjustment and tolerance, marked sensitivity, and insecure social and sexual impulses. This gives rise to a physiological and psychological tension which is relieved by alcohol. The alcoholic drinks to overcome embarrassment and self-consciousness and to get a release from his feeling of inadequacy. Davidson also believes that alcohol stimulates the sympathetic nervous system and that this may lead to some anxiety and fear. In the syndrome of

acute hallucinosis there is usually a prodromal feeling of inadequacy which is followed by anxiety, fear, agitation and derogatory hallucinations. The voices heard are usually of the same sex as the patient and may be multiple. Less often, olfactory, gustatory and visual hallucinations and ideas of poisoning are present. Several cases are cited where acute hallucinosis occurred without any history of abuse of alcohol. Heringa[6] described a case of acute psychosis with hallucinosis that occurred in an alcoholic during a period of abstinence. Claude[7] stated that the hallucination was the result of a profound modification of the personality of a person with a paranoid makeup which caused the invocation of an outside action. This modification might be caused by an affective shock, intoxication or an organic shock. The hallucinations represented the content of the fear and anxiety of the patient.

In order to add some further clarification to the subject of alcoholism and hallucinosis, the writer felt that it would be of interest to note the symptoms that occurred in psychotic episodes of alcoholics who had abstained from alcohol over a considerable period and to determine the nature and frequency of hallucinations in these cases. Such an experimental situation exists in the prison psychoses, or as they are officially designated, psychoses with psychopathic personality. As is well known, many prison inmates react to their incarceration with psychotic episodes. The symptoms shown during these episodes vary; they may include marked excitement, depression, hallucinations, ideas of reference, delusions of persecution and periods of confusion. A great number of cases of this type develop at the Dannemora State Hospital. The writer selected the last 50 of these among patients who were known to be intemperate and the last 50 among those who were believed to be relatively temperate and compared their symptoms with special reference to the presence or absence of hallucinations.

Among the 50 individuals who were relatively temperate, there were six only who were hallucinated in the auditory field. Of the other group, 38 had hallucinations during their psychotic episode. The hallucinations were usually accompanied by fear and anxiety and were often derogatory. Because of the environment, they frequently dealt with prison activities. They also often related to homosexual drives. This was partially due to the constitutional

predisposition of the patients and to the greater natural incidence of homosexual desires in such a monosexual environment as exists in a prison. The most frequent hallucination consisted of hearing others talking about the person, accusing him of being a "rat" (informer), and threatening to kill him. One patient heard them say, "Roast him! You'll get a shave." Some heard strange voices while others were sure that these sounds came from the inmates of adjoining cells. There was a great deal of name calling, including epithets such as "punk" (homosexual), "rat," "gorilla," "c. s.," and " s. o. b." Some also heard requests that they perform sodomy. The hallucinations were most frequent at night, which was probably due to the fact that during the daytime the patients were working and in contact with other inmates. At night, however, they were alone in their cells; after 9 o'clock the lights were out and they might lie awake receptive to fantasy in the dark. Although the hallucinations were similar qualitatively to those found in acute alcoholic hallucinosis, they differed quantitatively. They were not so severe or so persistent as those found after alcoholism. They usually subsided within about two weeks. Often the mere change of environment from the prison to the hospital was sufficient to make them disappear. They were much more dependent on the surroundings and more closely associated with paranoid trends than those occurring in acute alcoholism. A comparison of other symptoms in these two series—depression, mania, ideas of reference, delusions of persecution, mannerisms, neurotic trends and confusion—showed no appreciable difference between the two groups.

These data lead one to conclude that alcoholism has a close relationship to hallucinosis, but not in the sense that alcohol exerts a toxic effect which directly causes the hallucinations. It is more likely that the personalities developing hallucinosis are similar to those who are prone to imbibe alcoholic liquors excessively. The alcoholic is a socially and sexually insecure individual. He becomes intoxicated to overcome his feelings of inadequacy. Insecurity is also frequently associated with a paranoid makeup. Thus an overindulgence in alcohol may upset this type of personality and push him out of the world of reality, changing mere suspicion to projection and hallucination. Social and sexual inferiority are ag-

gravated by the prison environment. Paranoid trends can also have full play in such a milieu. In this case, the difficult environment may occasionally exercise an effect similar in some respects to that of intoxication, and the patient begins to project and hallucinate. The underlying instability of the patient, however, is the most important factor.

SUMMARY

Alcoholics who develop psychotic episodes while incarcerated, manifest hallucinations very frequently (39 of 50 cases) although they have been abstinent for periods varying from six months to 10 years. This is in contrast to nonalcoholics who develop prison psychoses. It is probable, therefore, that hallucinosis associated with alcoholism is related to the underlying constitution and personality rather than specifically to the alcohol.

Dannemora State Hospital
Dannemora, N. Y.

REFERENCES

1. Henderson and Gillespie: Textbook of Psychiatry, p. 274, Oxford University Press, London, England, 1932.
2. Schneider, C.: Studies on alcoholic hallucinosis, Psych. Bull., 9, p. 3, January, 1916.
3. Burns, G. C. H.: Alcoholism and manic-depressive insanity. St. Hosp. Bull., 7, p. 393, November, 1914.
4. Kirby, G. H.: Alcoholic hallucinosis, Psych. Bull, 9, p. 352, July, 1916.
5. Davidson, G. M.: The syndrome of acute hallucinosis, PSYCHIAT. QUART., 13, p. 466, July, 1939.
6. Heringa, S.: Atypical psychoses in alcoholic addicts, Psychiat. en. neurol. bl., 41, p. 47, January, 1937.
7. Claude, H.: Mechanism of hallucinations, PSYCHIAT. QUART., 4, p. 59, January, 1930.

INTRAVENOUS SODIUM AMYTAL MEDICATION AS AN AID TO THE RORSCHACH METHOD*

BY DOUGLAS M. KELLEY, M. D., KATE LEVINE, M. A., WILLIAM PEMBERTON, B. A., AND KATZ K. LILLIAN, M. D.

Soon after the introduction by Bleckwenn in 1930[1, 2] of sodium amytal as a therapeutic agent in the psychotic states, numerous workers[3-6] found that the intravenous injection of this drug in prenarcotic doses produced transient ameliorating effects in certain psychotic patients. The mental changes which resulted were for the most part striking shifts from withdrawn, resistive, uncooperative attitudes to communicative, cooperative, friendly ones. Because of these findings it seemed reasonable to assume that patients who, during the course of a study by means of the Rorschach method, refused so many cards that accurate personality description or diagnosis was impossible, might, under the influence of prenarcotic doses of sodium amytal, give a sufficient number of responses to permit an adequate evaluation of the record.

Procedure

In studying the cases a preliminary Rorschach test was made in the customary manner as advocated by Klopfer.[7] Sodium amytal in a freshly prepared 10 per cent solution was next injected intravenously at a rate not to exceed 1 cc. a minute. Signs of mental amelioration generally appeared after the administration of about 3-4 cc., but usually the best results were obtained with 5-6 cc. of the drug. Patients vary considerably in their reactions to the medication; it is imperative not only that a sufficient amount of sodium amytal be given to induce the necessary freedom from inhibition, but on the other hand that excessive amounts be avoided so that the administration and inquiry of the Rorschach method may be completed before the patient becomes too drowsy to cooperate. Rapid injection seems to give no advantageous reactions; in addition, it introduces the increased hazard of a possible depression of the respiratory centers.

*From the department of psychiatry, New York State Psychiatric Institute and Hospital and the department of psychology, Rockland State Hospital.

As soon as the patient showed increased relaxation, friendliness and willingness to cooperate, the injection was terminated and the Rorschach study begun. It is important to complete the test in as short a time as possible so that the patient does not become too sleepy. Also, careful observations of any changes in the patient's state of consciousness should be noted, later to be correlated with the responses themselves.

The patients included in this study were selected from the routine hospital population, in most cases because of their refusal to respond to routine Rorschach administration.

Results

Table 1 presents the data pertinent to the cases studied, their clinical diagnosis, the preamytal Rorschach diagnosis and number of card rejections, and the amytal Rorschach diagnosis and number of rejections.

The qualitative change in the response is as important for proper evaluation as the actual increase in responses. This improvement cannot be so easily shown in tabular form, but a few typical examples will clearly illustrate the qualitative response change under sodium amytal medication. In case 3, the four responses given were of little value as they were all "spine" or "backbone." Under amytal, 20 scorable responses were given with no rejections and a diagnostic picture was obtained. In case 2, only five responses, three of which consisted of only description or color-naming were given, and differentiation between an organic process or dementia præcox was impossible. Under amytal the 12 responses to nine cards portrayed a clearcut type of dementia præcox. In case 9, the value of the qualitative aspect is clearly seen, for while the diagnosis of dementia præcox was obvious from the 10 initial responses, further differentiation could not be made except with the amytal record. Since this case is a fairly typical one, the original responses showing scoring difficulties will be given followed by the scoring tabulations of the responses under amytal medication, the actual responses of this record being of little interest to us here.

TABLE 1

				Preamytal Rorschach findings		Postamytal Rorschach findings	
Case	Age	Sex	Clinical diagnosis	Reject	Diagnosis	Reject	Diagnosis
1	28	F	Psychoneurosis (anxiety hysteria)	8	Not possible	0	Psychoneurosis (anxiety hysteria)
2	22	F	Dementia præcox hebephrenic	5	Not possible	1	Dementia præcox, hebephrenic
3	51	M	Manic-depressive psychosis	6	Not possible	0	Depression, probably manic-depressive
4	54	F	?Dementia præcox, catatonic; or of ?organic etiology	10	Not possible	2	Organic psychosis
5	21	M	Dementia præcox, catatonic	10	Not possible	3	Dementia præcox, catatonic
6	21	M	Dementia præcox, catatonic	9	Not possible	0	Dementia præcox, catatonic or simple
7		F	Dementia præcox, catatonic	10	Not possible	2	Dementia præcox, catatonic
8	28	F	Psychosis with multiple sclerosis	5	Questionable organic psychosis	1	Organic psychosis
9	30	M	Dementia præcox, hebephrenic	0	Dementia præcox (? as to type)	0	Dementia præcox, hebephrenic with paranoid trends
10	10	F	?Dementia præcox, hebephrenic	0	?Psychoneurosis	0	Psychoneurosis (anxiety hysteria)
11	29	F	Psychoneurosis (anxiety state)	0	Psychoneurosis (anxiety state)	0	Psychoneurosis (anxiety state)
12	38	F	Dementia præcox, catatonic	10	Not possible	0	Dementia præcox, paranoid with catatonic features
13	35	M	Dementia præcox, catatonic	10	Not possible	0	Dementia præcox, catatonic
14	33	M	Dementia præcox, paranoid	5	Not possible	0	Dementia præcox, paranoid

Card	Time—Seconds	Response
I.	50	May be the medicinal or psychological aspect. May be the aspiration toward the sublime and divine. May be the biblical aspect of offending the natural sphere of things.
	135	
II.		The natural conflict of the order over the offense of the sphere
	65	of things. Beaver furskin—process of manufacture.
III.	25	The planet aspect in relation to human ideologies in the realm of the social or biological.
IV.	50	This is still the Freudian aspect. No, I think Jung's.
V.		A mark in space, but it has a color reaction on the mind. It may be meaningless, but everything has a reaction on the biological. Has a reaction to the mind in different climates.
VI.	65	On the different shades of shadows. May be stalactite or stalagmite reaction.
VII.	15	Don't know. May be chemical map compound.
VIII.		A repetition of impressions. Changes of phobias.
IX.		Unanimity of thought and of emotions—of thought as related to plants, earth and man.
X.		Artistic esthetic image from the primitive to the cultural.

This patient after having been given 7½ gr. of sodium amytal showed no appreciable change in his behavior but gave Rorschach responses which when scored were as follows:

Total R = 27
Total T = 24 min.

	W	12	M	2			
	D	13	FM	2		H	2
	S	1	m	0		Hd	0
	dS	1	k	0		A	13
		—	K	1		Ad	0
		27	FK	0		A obj.	1
			F	12 (3)		Ntr.	4
			Fc	0		Obj.	2
P = 3			c	3		Plant	1
O = 6			C'	1		Ptg.	2
			FC	2		Fire	1
			CF	3		Cloud	1
			C	1			—
				—			27
				27			

From the above tabulation the qualitative changes can easily be seen, and a definite diagnosis and detailed personality description is possible from the second record.

Discussion

From a quantitative point of view, it is obvious that in all the cases studied which originally showed rejection of five or more cards, the use of sodium amytal permitted the scoring of a record with much fewer rejections. It is possible of course that certain patients will not show this reaction, and one of those here studied (case 5) on the first amytal administration continued mute. Repetition of the test under amytal the following day, however, produced a usable record. The technique should be repeated in this type of case. Most of the original Rorschach records as seen from the chart were inadequate for use as diagnostic aids because of a paucity of responses; this difficulty was completely overcome in all cases by the use of sodium amytal.

From a qualitative point of view, our ideas must still be mostly theoretical. The pharmacological action of sodium amytal is still not definitely known and numerous hypotheses have been suggested to explain its action. The drug certainly seems to show some definite effect upon the autonomic centers in the brain stem (as demonstrated by Gildea, Himwich, Hubbard and Fazikas[8]) and also seems to act as a physiological cortical depressant, diminishing normal inhibitory action of the cortical cells (as postulated by Herman[9]). Herman also believes that "with the removal of inhibition and the effect on the autonomic centers there is produced an easy path for the translation of emotional reactions and patterns into speech and action."

Clinically, most workers agree with Thorner[6] that "the changes observed in patients to whom sodium amytal was administered intravenously were largely in the nature of a psychic release," and Lindemann[5] found in all his cases "a striking change from a resistive, seclusive attitude to friendly and emotionally warm communication." These findings are borne out by the quantitative improvement in the Rorschach responses, and the qualitative changes conform with Lindemann's statement that, "The abnormal thoughts hidden before were communicated after the drug administration."

As can be seen from the table, diagnosis was facilitated by both the quantitative and the qualitative changes, and apparently the amytal itself produced no other change in the patient's Rorschach responses than to permit a free flow of basic responses. It is of

course important to know whether sodium amytal itself is capable of influencing the Rorschach responses. However, since the amytal diagnoses all checked accurately with the clinical diagnoses (except in cases 4 and 10, where clinical diagnoses themselves are still questionable) it seems reasonable to assume that changes, if there are any, due directly to the sodium amytal, are of little significance in diagnostic interpretations. Further work upon normal individuals is being undertaken, however, to determine accurately this point and also to check certain assumptions in the literature that the action of sodium amytal in prenarcotic doses is similar to that of moderate doses of ethyl alcohol.

Summary

1. The use of intravenous sodium amytal in prenarcotic doses as an aid to the Rorschach method is described.

2. Following sodium amytal injections, the Rorschach responses show a greater number of responses and fewer rejections of cards, making diagnosis possible in cases previously considered unavailable.

3. The responses under sodium amytal medication are qualitatively less bizarre and stereotyped and permit far finer nuances of personality description.

4. The need for further experimental work on normals is stressed.

REFERENCES

1. Bleckwenn, W. J.: Production of sleep and rest in psychotic cases. Arch. Neurol. and Psychiat., 24: 365, 1930.
2. Bleckwenn, W. J.: Narcosis as therapy in neuropsychiatric conditions. J. A. M. A., 95: 1165, 1930.
3. Harris, M. M., and Katz, S. E.: The effect of the administration of sodium amytal and sodium rhodanate on mental patients. Am. Jour. Psychiat., 12:1065, 1932.
4. Harris, M. M., Horwitz, W. A., and Milch, E. A.: Regarding sodium amytal as a prognostic aid in insulin and metrazol shock therapy of mental patients. Am. Jour. Psychiat., 96: 327, 1939
5. Lindemann, E.: Psychological changes in normal and abnormal individuals under the influence of sodium amytal. Am. Jour. Psychiat., 11:1083, 1932.
6. Thorner, M. W.: The psychopharmacology of sodium amytal. Jour. Nerv. and Ment. Dis., 81:161, 1935.
7. Klopfer, Bruno: Theory and technique of the Rorschach performance. Rorschach Research Exchange, 2:1, 1937.
8. Gildea, E. F., Himwich, H. E., Hubbard, A. E., and Fazikas, J. F.: A comparative study of some of the changes produced by various types of drugs in schizophrenic patients. Am. Jour. Psychiat., 91:1289, 1935.
9. Herman, Morris: The use of intravenous sodium amytal in psychogenic amnesic states. Psychiat. Quart., 12:738, 1938.

THE EYE GROUNDS IN FUNCTIONAL PSYCHOSES GIVEN INSULIN-SHOCK THERAPY*

With a Review of the Literature

BY ALEXANDER GRALNICK, M. D.

This study was undertaken with the hope that its findings would prove of value to both the psychiatrist and ophthalmologist. The use of insulin in the armamentarium of the psychiatrist, with its occasional damaging physical consequences to the central nervous system, makes the eye an object of interest. The retina not only is derived from the same germ-layer as the brain but also possesses a similar histologic structure with cells analogous to those injured in the brain. The uncertainty of our knowledge of what occurs in brains of patients surviving insulin-comas and convulsions makes a fundus-study desirable, for it may give a clue to what occurs in the organ from which the eye develops.

When large doses of a drug are administered, it is customary to investigate organs likely to be affected. Thus, treatment with arsenicals demands study of vital organs, including the eye, to forestall disturbance of intrinsic function and structure. Similarly, there is reason to follow the fundus during insulin therapy in search of possible changes. As liver-function is studied to prevent over-zealous treatment with dangerous drugs, so might the fundus be observed for signs of serious consequences to come. In addition, the retina might act not only to warn of mishap, but also to show prognostic criteria.

Not a few ophthalmologists think that insulin used therapeutically contributes to the diabetic fundus picture. Thus, Poyales[1] says that diabetic fundus-lesions grow worse and tend toward a hemorrhagic type when insulin treatment is instituted. Some think, too, that insulin plays a part in the production of retinal detachment. A therapeutic procedure which administers thousands of units of insulin in a short time could enlighten us on these interesting ophthalmological problems. Finally, from the point of view of pure scientific curiosity, which leads to the accumulation of

*Presented at the Interhospital Conference, New York State Psychiatric Institute and Hospital, New York City, April 18-19, 1940.

knowledge, we may consider it valuable to observe and report on the fundi of psychotic patients who have had insulin-shock treatment.

Review of the Literature

A. *The Fundus of Mental Patients:* As far back as 1856, the fundus of the mentally diseased was an object of study. In that year Ludwig[2] posed the question: "Can we judge the brain by an examination of the retina of mental patients?" Though he could not answer, he admonished the psychiatrist to study the eye seriously. Since then, not a few examinations of fundi have been done, with much difference of opinion as to findings. Wollenberg,[3] in a study of 847 men and 956 women with functional psychoses, found 1.2 per cent of the former and 0.72 per cent of the latter to have congenital anomalies, including defective papillae, myelinated nerve-sheaths and vessel anomalies. Although other psychoses were studied as well, no definite diagnostic criteria were discovered in the 6,400 patients he examined; and the cases seen were not well classified into groups.

Uhthoff[4] in 1915 reported that in schizophrenia there is no characteristic fundus-picture. He disagrees rather sharply with Tyson and Clark,[5] who studied 109 cases of dementia præcox and divided the fundus-pictures into three groups: (1) Congestion of discs; hyperemia and edema; dilated, dark colored veins; slightly contracted arteries and blurring of the edges of the discs. These changes constitute, according to them, a low-grade perineuritis of the optic nerve. (2) Congestion of the nasal side with temporal pallor of the discs, dilated veins and contracted arteries. (3) Pallor of the discs, dilated veins and contracted arteries. These changes constitute anemia and partial atrophy of the optic nerve.

Tyson and Clark declare that more marked changes are found in the more rapidly deteriorating types of schizophrenia. Further, they say the optic nerve lesions described are in accord with "our best knowledge of the pathologic anatomy of dementia præcox." They offer these "eye-syndromes" as of diagnostic and prognostic value.

The preponderance of opinions controverts their findings and conclusions. Their optimism seems to be as well founded as their

presupposition that schizophrenia is an autotoxic disease that principally injures vessels; and it is likely that their findings were influenced by their original thesis of the etiology. Wintersteiner[6] denies that there are vascular changes in the fundi of schizophrenics, but in his study of 120 cases of dementia præcox he reports some changes in small percentages. Though he does not indicate clearly whether they are chance complications, one gathers that he agrees with Uhthoff[4] rather than with Tyson and Clark.[5]

Kaminskaja-Pavlowa,[7] after studying 143 purely functional cases, agrees with Uhthoff and adds that change of the fundus neither parallels the severity of the mental illness nor progresses with the disease's advance. Holmes[8] found 20 per cent of 2,000 psychotic patients to have demonstrable fundal pathology related to the etiological factors responsible for their psychoses. However, these had organic psychoses or organic diseases associated with the functional psychoses. The remaining 1,600 cases were not discussed, but it may be inferred that they showed no pathology. Unfortunately they were not grouped.

Though the literature gives some conflicting evidence, there is nearly a consensus that the functional psychoses do not present characteristic eye grounds. Changes are incidental and cannot logically be attributed to the illnesses. It is certain that changes do not progress with the course of mental illness.

B. *The Effects of Insulin, Pertinent Pathological and Physiological Considerations:* It is generally conceded from experimental work and from study of human autopsy material that insulin-coma produces cerebral damage. The extent and mechanism are a matter of debate. Weil[9] finds that the brains of rabbits dying in hypoglycemic convulsion show diffuse severe damage of the ganglion cells, while those allowed to survive show less marked damage over a wide area. His rabbits remained free of changes if 70 or less units were injected over a period of two months, whereas those receiving 200 to 400 units had severe changes. Reed and Dancey,[10] and Accornero[11] agree that the cerebral damage is diffuse in animals and humans dying in convulsion. The former, however, think that if glucose is given soon enough there is no permanent change, whereas Accornero says that animals sacrificed two to 15 days after 30 insulin shocks show less diffuse damage and more lo-

calized areas of necrosis related to circulatory disturbances. Ferraro[12] and Malamud[13] also stress the diffuse nature of the cerebral damage and point out that the focal changes are merely local accentuations of the generalized process. Roizin[14] believes that the "toxic-metabolic process" caused by hypoglycemia produces diffuse cerebral changes, but, with Ferraro, holds the focal changes to be due to vascular disturbances, including increased permeability and vessel-wall changes.

Although there exists difference of opinion as to the physiological mechanism responsible for cerebral pathology, it is principally thought that in the presence of hypoglycemia the nerve cells cannot utilize oxygen, and therefore degenerate. Weil[9] says the cause is an intracellular anoxemia, due to the presence of large doses of insulin, not a vascular disturbance. Dameshek,[15] Gellhorn[16] and Wortis[17] agree that a decreased oxidative process plays an essential rôle in the production of the clinical and neurological symptoms. Malamud[13] interprets the brain-degeneration as due to a direct toxic effect of insulin on the parenchyma. Ferraro[12] lends him a measure of support but goes one step further and, with Grayzel[18] and Accornero,[11] thinks that a vascular mechanism is important. The latter two stress the importance of the convulsion itself. Grayzel thinks minimal or no changes occur in animals unless convulsions are severe and prolonged enough to produce anoxemia due to circulatory disturbance. Accornero says cerebral changes are more severe if convulsions occur.

C. *Appearance of Fundus and Histopathology of the Eye:* Ciotola[19] reports the fundus in hypoglycemic coma to be paler than normal. This he attributes to a decreased blood-flow to the choroidal plexus. Cozzoli[20] and Accardi[21] say insulin produces hyperemia of the retina, while Wolf and De Jongh[22] report negative findings in hypoglycemic rabbits.

Microscopic study of the eyes of rabbits dying in hypoglycemic convulsions has been reported in only one paper discoverable in the literature. Mamola[23] finds that there is nothing of note except small hemorrhagic foci near the ciliary body. These he attributes to the convulsions rather than the insulin coma.

D. *Intraocular Tension:* Though hypoglycemia may affect the seeing apparatus in various ways, producing visual disturbances

and pupillary changes, (Rynearson[24]) the preponderance of study has been on intraocular tension. That decreased intraocular tension frequently exists in diabetic coma is generally accepted, but Patek's[25] statement that "hypotonia bulbi is not found in comas other than those of diabetic origin" finds a different reception. Weichmann[26] says that humans show a reduced intraocular tension in insulin hypoglycemia. Ciotola[19] studying 11 insulin-treated psychotics, found generally a decrease in tension down to 12 mm.Hg. He never found a hypertonia bulbi. Vestergaard[27] finds that both normal and diabetic individuals show a fall in intraocular tension of 17.5 per cent beginning 10 to 20 minutes after insulin injection.

Working with rabbits, Mamola[23] and Wolf and De Jongh[22] find that if sufficient insulin is given, ocular tension is decreased. However, the former says that the sugar content of the blood is not of importance and that the tension remains lowered for 24 hours, whereas the latter report that glucose given soon enough after convulsions will usually prevent hypotonia bulbi. Interestingly enough, Wolf and DeJongh say that the tension is lowered only after a convulsion and that strychnine, camphor and picrotoxin in convulsing doses also produce decreased ocular tension. Raiha[28] reports a decreased intraocular tension in rabbits followed by an increase of a transitory nature. Gray[29] says that at the beginning of "insulin coma" there is little change in the intraocular tension, but in repeated reactions, e. g., occurring every 24 hours, there is a gradual "hardening of the eyes."

Richter,[30] Bistis[31] and Accardi,[21] report that insulin in sufficient dose will raise the intraocular tension. Richter states that the effect endures six hours and the rise may be to 38 mm.Hg. Cossa,[32] finds that in hypoglycemic coma the diastolic pressure of the retinal artery doubles.

The mechanism whereby this fall in tension occurs is explained in various ways. Weichmann[26] and Ciotola[19] think that concentration of the blood due to sweating, with consequent increased colloidal osmotic pressure, is the chief reason for lowered tension. Ciotola gives as a minor reason the accumulation of substances with capillary toxicity. This seems to gain confirmatory support from Wolf and DeJongh's[22] finding that serum from convulsed ani-

mals (animals having convulsions) produces decreased intraocular tension when injected intravenously into other rabbits or dogs. They believe they have separated out of it the responsible agent. Mamola[23] thinks that ocular tension is lower because of a hypotensive principle present in insulin, which operates regardless of the administration of glucose.

E. *Insulin and Retinal Detachment:* In the past 10 years there have been reported two cases of retinal detachment supposedly caused by insulin administration. Alperin[33] describes the case of a diabetic being treated with relatively small doses of insulin suddenly becoming blind after an injection. Examination revealed detachment about the macula. He attributes this to disturbed electrolytic and osmotic processes in the blood and ocular fluids which result in fluid being thrown into the subretinal space from the choroidal plexus of vessels. McBean[34] reports blindness due to retinal detachment and secondary iridocyclitis in a 35-year-old hyperopic woman who received 10 units of insulin every day for two months to improve her appetite. He ascribes her blindness to the insulin.

Technique of Study

Because the cases observed were not under the writer's immediate care, examination of fundi while patients were in coma was precluded. Examination of patients brought to the hospital clinic was possible, however. The treatments were given every weekday morning; and the examinations were made Friday afternoons or Saturday mornings. As a rule each subject was seen every second week, in some cases every third. Inasmuch as the primary interest was in possible permanent fundus-changes, this course seemed satisfactory as well as practical.

Each time a patient was seen, blood pressure was recorded; and a urine sample was examined for sugar and albumen when one could be obtained. In each case at least one urine specimen was thoroughly examined; and in most, several examinations were made, so that accurate urinary status was determined. Wide dilatation of the pupil was obtained at every visit with the use of

2 per cent homatropine so that a thorough search of the fundus could be made each time. After examination, 1 per cent pilocarpine was instilled in the eye.

The number of times each patient was seen varied. Some patients were paroled immediately after completion of therapy, and some were at times too uncooperative for examination. In addition, the work was started by seeing patients who had just completed treatment. Generally, however, they went through the examination outlined about six times, decreasing greatly the possibility of error.

Of the 35 patients so studied, 21 were seen once before treatment was begun, several times during the course of treatment, and for a period afterward ranging from one day to 10 weeks. Each of these was seen between four and 11 times. Five of these were seen only once following treatment, one day after therapy, as they were paroled immediately. Other cases were followed, as planned, for eight to 10 weeks after completion of the insulin course. Fourteen patients were first observed after treatment was discontinued. These were seen from two to eight times each and were followed for from one to 16 weeks, an average of seven weeks. One was followed for 16 weeks and seen eight times because of an interesting fundus finding.

The 35 patients were treated from six to 12 weeks, and they ranged from 20 to 55 years old. The number of insulin treatments, which each had, varied from 27 to 58, with the lowest total dosage 840 units and the highest 6,165 units. The minimum total hypoglycemic hours a patient endured was 113, the maximum 254. In her entire course of treatment, one patient only once became semicomatose, while another had 40 real comas. Insulin convulsions varied from one to 11. In five cases, metrazol was given in addition to insulin therapy.

Treatment in all the cases was terminated because the administrator deemed it sufficient in amount. It resulted in 10 considered much improved, three improved and 22 unimproved. Thirty-one of the cases were of the dementia præcox group, three manic-depressive and one psychoneurosis. All had normal urinary findings; and the blood pressures ranged from 90/64 to 150/100, the great majority being within normal limits.

The possibility of fundus-change showing many months after discontinuance of treatment induced further examination. Thirty-five cases where insulin injections had terminated five to 34 months previously were taken at random. The subjects had remained at the hospital and were available for examination with the technique used previously. Each was seen only once.

These latter patients ranged in age from 19 to 42. The shortest period of treatment had been five weeks and the longest 19 weeks, with the number of individual treatments varying between 22 and 89. The lowest total dosage given any one patient was 745 units, and the highest 11,125 units. The minimum total hypoglycemic hours a patient endured was 89, the maximum 404. One patient became comatose only twice; another became so 51 times. Insulin convulsions varied from none to seven.

In most cases treatment in the second group was terminated because the administrator deemed it sufficient in amount. In two, acute illnesses ended the therapy. The treatment resulted in 26 unimproved, five improved, and four much improved cases. Thirty-four of the patients had been classified in the dementia præcox group; and opinion in the thirty-fifth case was divided between schizophrenia and postencephalitic psychosis. All had normal urinary findings; and the blood pressures ranged between 100/60 and 160/110, the great majority within normal limits. One received metrazol as well as insulin therapy.

Findings

Of the first group of 35 patients who were seen before, during and after treatment, 31 had fundi essentially within normal limits. Two had fundus changes which were found before treatment was started and which did not progress during the therapy. One of these patients was 55 years old and had changes characteristic of moderate arteriosclerosis. The other was a 35-year-old woman who had an old chorioretinal degenerative process just temporal and superior to the right macula. According to relatives, "Her eyes were always weak." On admission, her vision was, L.10/10, R. 10/40. Ten weeks after treatment, her vision was the same. The thirty-fourth patient was a 23-year-old female who showed bilat-

eral temporal pallor of the discs seven weeks after treatment was completed. The last of this group was a 22-year-old girl first seen immediately after completion of seven weeks of insulin therapy. At this visit an extensive, healed chorioretinal degeneration at the periphery of the left fundus was found. The girl was seen eight times in the course of 16 weeks, without any change in the lesion being detected. Anamnesis disclosed that she had been in an automobile accident two years before, suffering a concussion with accompanying visual disturbance of this eye.

In the second group, 30 of the 35 patients had essentially normal fundi. A 24-year-old man, who had had eight weeks of treatment with two convulsions, showed eight months after treatment, a left disc paler and sharper than the right. A 42-year-old woman had fundi essentially negative except for a slight degree of arterial spasm. She was seen 31 months after treatment. A woman of 39 showed a high degree of myopia and a small temporal crescent of the left fundus 26 months after treatment. A 36-year-old man, who had been treated for seven weeks, was seen 22 months after therapy. His right fundus had a pin-head sized, clearly-defined pigment spot just nasal to the disc. The surrounding retina was normal as were the rest of his eyegrounds. A 33-year-old female, seen 29 months after treatment, had similar findings. In the right fundus, there were two such spots, in the left, one. These were along the course of arteries.

In the past nine months, the writer has had the good fortune to see two cases of prolonged coma. Both had been diagnosed dementia præcox, paranoid. The first occurred in a 46-year-old man. On the third day of his treatment, 35 units of insulin were given; and he went into a coma that lasted 36 hours. During this period, examinations were negative except for vascular changes attributable to his age and elevated blood pressure. Treatment was discontinued. A month later, his fundi showed no essential change. He developed pulmonary tuberculosis. When seen nine months later, his fundi showed no further changes.

The second case of prolonged coma occurred in a 40-year-old woman who died after remaining unconscious for 10 days. She was examined several times a day but showed no fundus changes, except some venous engorgement in the last two days. She had

been treated with insulin for eight weeks and had been receiving 125 units daily for several weeks before her final coma. On several occasions, intravenous glucose, as well as intranasal sugar-solution, had to be given to interrupt coma. She had 25 comas but no convulsions. She received 2,500 units of insulin in all.

Summary

Though there is some disagreement, there is virtually a consensus that the functional psychoses neither present characteristic eyegrounds nor fundus-changes that progress with the advance of mental illness. Positive findings are associated with other pathology of an organic nature, but the incidence is no greater than in normal persons. Given no generalized physical process or local eye-disease, the appearance of the fundi in psychotic individuals will be as in the mentally normal.

It is generally conceded that insulin in doses sufficient to cause hypoglycemic coma and convulsions in experimental animals will produce diffuse cerebral damage, with accentuated localized areas of destruction. In humans, prolonged, fatal coma produces much the same result. The similarities between the retina and the brain make the accessible fundus a desirable region to study.

The literature differs regarding the appearance of the fundus during insulin coma, with both hyperemia and anemia reported. One histologic study of the eyes of a rabbit dying in hypoglycemic convulsion shows minor hemorrhagic foci near the ciliary body. The intraocular tension during insulin coma has been reported by some to be decreased and by others to be increased. Ophthalmologists declare that diabetic fundus changes are aggravated by insulin; and two ascribe retinal detachments to insulin-administration.

No report of the human eye-grounds after insulin-shock therapy has been made previously. This study is meant to fill that void. Two groups of 35 cases each were studied with the ophthalmoscope. Most members of the first group were seen just before therapy, as well as during it; and observations ranged up to a period 16 weeks

after treatment. This series was studied for immediate changes. The cases of the second group had been treated five to 34 months before being seen. This series was studied for possible late changes.

Conclusions

1. In an ophthalmoscopic study of the eye-grounds of 70 patients with functional psychosis, who received insulin-shock therapy, no typical changes were found which could be attributed to the treatment. Retinal detachment occurred in none of the cases.
2. There is general agreement as to the lack of any characteristic appearance of the fundi of patients with functional psychosis.
3. The duration of a functional psychosis does not influence the appearance of the fundus.

Appreciation

The author wishes to express his appreciation to Harry McGrath, M. D., F. A. C. S., Marcus Schatner, M. D., and Reidar Trygstad, M. D., for their valuable aid and cooperation.

Central Islip State Hospital
Central Islip, N. Y.

REFERENCES

1. Poyales: Insulin in ophthalmology. Arch. d'ophth, 1924.
2. Ludwig, G.: Allg. Zeitschr. f. Psychiatrie. Bd. XIII, H. 1, S72, 1856.
3. Wollenberg: On congenital anomalies of the eyes in psychosis. Charité Annalen XIV, S. 470, 1889.
4. Uhthoff, W.: Graefe-Samisch—Handb'k d. ges. Augenheilk Bd. IX, Abt. 2B, S 1560, 1915.
5. Tyson, H. and Clark, P.: The eye-syndrome of dementia præcox. (a) J. A. M. A., May 2, 1908. (b) Arch. of Ophth. Vol. 41, No. 3, Pg. 223, 1912.
6. Wintersteiner, H.: Diseases of the fundus in psychosis. Zeitschr. f. Augenheilkunde, Vol. 23, Pg. 1 and 124, 1910.
7. Kaminskaja-Pavlowa and Goldblatt: Eye-findings in mental disease and epilepsy. Allg. Ztschr. f. Psychiatrie, 89:62-70, July 6, 1928.
8. Holmes, W.: Eye grounds in mental disturbances. Ohio State M. J., 34:765-67, July, 1938.
9. Weil, Liebert and Heilbrun: Arch. Neurol. and Psychiat. 39:467-81, March, 1938.
10. Reed and Dancey: Insulin therapy of dementia præcox with statistical data. Internat. Clin., 2:101-125, June, 1938.
11. Accornero, F.: Exptal Histopathologic Researches on Shock, Am. J. Psychiat. (supp.) 94:130-33, May, 1938.
12. Ferraro, A., and Jervis, G.: Brain pathology in four cases of schizophrenia treated with insulin. Psychiat. Quart., Vol. 13, April, 1939.
13. Malamud, N., and Grosh, L.: Hyperinsulinism and cerebral changes: report of a case due to islet cell adenoma of pancreas. Arch. Int. Med. 61:579-99, April, 1938.
14. Roizin, L.: Histologic alterations of the central nervous system in animals subjected to insulin shocks. Rivista di Patologia nervosa e mentale, Vol. 53, Pg. 491, May, 1939.

15. Dameshek, Wm. et al: Mechanism of the neurologic symptoms in insulin hypoglycemia. Arch. Neurol. and Psych. 33:1-18, January, 1935.

16. Gellhorn, E.: Action of insulin hypoglycemia on the central nervous system. J. A. M. A., 110:1433-34, April 30, 1938.

17. Wortis, S.: Effects of insulin on brain metabolism. N. Y. S. J. Med. 38:1015-21, July 15, 1938.

18. Grayzel, D.: Changes in the central nervous system resulting from convulsions due to hyperinsulinism. Arch. Int. Med. 54:694-701, November, 1934.

19. Ciotola, G.: Behavior of ocular tonus in insulin coma. Boll. d'Ocul. 17:738-54, September, 1938.

20. Cozzoli, G.: Cited by Mamola. Tip. Cecchini, Roma, 1923.

21. Accardi: Cited by Mamola. Boll d'Ocul IV, Pg. 289, 1925.

22. Wolf and DeJongh: Concerning a substance (insulin) which reduces intraocular tension. Klin. Wochenschr., XLI, Pg. 344, 1925.

23. Mamola, P.: Effects of insulin on ocular tension. Riv. san Siciliana, 21:431-37, March 15, 1933.

24. Rynearson, E.: Neurologic manifestations of hyperinsulinism. J. A. M. A., 103:1191-99, October 20, 1934.

25. Patek, A.: Soft eyeball in diabetic coma. J. A. M. A. 92:438-40. February 9, 1929.

26. Weichmann, E.: Difficulties in diagnosis of insulin coma. J. A. M. A. 92:1495-96, May 4, 1929.

27. Vestergaard, J.: Insulin and ocular tension. Acta. Ophth. 7:273-300, 1929.

28. Raiha, C., and Gronlund, L.: Effects of insulin on intraocular tension. Skandinav. Arch. f. Physiol. 62:70-80, July, 1931.

29. Gray, W. A.: Brit. Journ. of Ophth. XVII, Pg. 577, 1933.

30. Richter, A.: Insulin and ocular pressure. Klin. M. f. Augenh., v 76, Pg. 835. June, 1926.

31. Bistis, J.: Effects of insulin on ocular tension. Arch. d'ophth, 49:375-8, June, 1932.

32. Cossa, et al.: Hypoglycemic coma and retinal arterial tension. Rev. Neurologique, 69: Pg. 714-20, June, 1938.

33. Alperin, D.: Retinal detachment complicating insulin therapy. Am. J. Ophth. 12:486, 1929.

34. McBean, G.: Retinal detachment following administration of insulin. Am. J. Ophth. 12:825, 1929.

METRAZOL SHOCK THERAPY

Report of a Case with Activation of a Latent Diabetes

BY IRVING GREENFIELD, M. D.

A valuable approach in the management of psychiatric problems was at hand when von Meduna,[1] in 1935, published his observations following the study of a group of patients who had received metrazol shock therapy. Four years later, von Meduna and Friedman[2] reviewed the literature and summarized the complications which were recorded following the widespread use of this type of therapy. Since then, additional complications have been reported.

At first, this form of treatment was limited to that group of patients who showed schizophrenic tendencies. More recently, metrazol has been employed in private sanataria and in general hospitals having psychopathic wards, for the purpose of shortening prolonged manic depressive attacks.[3] Now that its field has been enlarged, the increased frequency with which it will be employed will offer a greater opportunity to study its untoward reactions.

The protocol of a patient who developed an aplastic anemia as a complication of metrazol treatment was described by Epstein.[4] Wender and Epstein[5] noted disturbances in the reticuloendothelial system following the use of metrazol. Disturbance of sugar metabolism, not previously recorded as a complication of metrazol therapy, forms the basis of the following case report.

Case Report

B. C., a 37-year-old white female, had been under observation for about three years prior to the onset of complaints referable to her present illness. She was one of seven children and was of a worrying disposition. The responsibility of the home always rested more heavily on her shoulders than on any of the other members of the household. At 35, she married a man who was impotent; and, though outwardly she accepted the misfortune without disturbance, on several occasions she manifested anxiety concerning the fact that she had never been able to take part in coitus.

The patient was a fine executive and held a responsible position in a large department store. About March of 1940, there was a change in managerial personnel, and the patient was asked to resign her position because of what she termed "personal reasons." This caused her great grief. In April, she complained of feeling very tired. Her husband noticed that she showed a lack of interest in herself and was depressed. She no longer cared about her clothes and was totally disinterested in her environment. Though she remained in bed most of the time, she continued to complain of fatigue almost to the point of exhaustion. She could not concentrate on any problem long enough to attempt its solution. Problems which she recognized as inconsequential in importance assumed tremendous proportions and caused distress.

She worried considerably concerning her condition, her finances, and the health of the members of her family. She was nervous and apprehensive. Often she spoke of "the futility of it all." She was sure she could not recover and felt a certain amount of pain because of the distress she was causing her husband and also because of the financial burden she had become to him. She would listen attentively to any attempt to cheer her up or to any therapeutic recommendation which was made and then, shrugging her shoulders, would ask, "What's the use?" During the month of April, she was seen on several occasions, and a diagnosis of retarded depression was made. Urinalyses on four occasions during this period of observation were negative.

The patient was finally referred for ambulatory metrazol shock therapy. Following the sixth such therapeutic treatment, she showed a remarkable improvement. Her mental attitude had changed completely. Her entire philosophy of life had changed; and she was aware of "the worthwhileness of it all."

At about this time, her husband noted that her appetite had increased tremendously and that she had begun to have polydypsia and complained of polyuria. A routine urinalysis revealed 3 per cent of sugar and the patient was admitted to the hospital for investigation. The results of the laboratory studies were as follows: The admission specimen of urine contained 1 per cent of sugar and was negative for pathological elements. Wassermann and Kline reactions of the blood were negative. The blood chemi-

cal values expressed in mgm. per 100 cc. of serum were: sugar—242 mgm., urea—24.5 mgm., chloride—336 mgm. The carbon dioxide combining power was 62.2 volumes per cent, potassium 16.3 mgm. per cent, sodium 320 mgm. per cent, total cholesterol 247 mgm. per cent, combined cholesterol 54 mgm. per cent, free cholesterol 22 mgm. per cent.

Ten days after her admission, the patient was discharged from the hospital. Her diet contained 250 grams of carbohydrate, 100 grams of protein, and 60 grams of fat. She required 60 units of regular insulin daily. Since the time of her discharge from the hospital she has remained on the diet noted. Several attempts to cut down the total caloric intake have been unsuccessful, because the patient complained of hunger about two hours after meals. She has remained controlled; and only on occasions when she found it impossible to endure the gap between meals without food have the urine specimens been found to reduce Benedict's solution. Protamine zinc insulin has been substituted for regular insulin. At the present time, the patient is gainfully employed. She requires five grains of thyroid extract daily. Recent investigations reveal a basal metabolic rate of plus one and a blood sugar level of 131 mgm. in 100 cc. of whole blood. The urine is sugar free.

Her past history is of extreme interest and, therefore, is recorded in some detail. The patient came under observation for the first time in 1937, when she complained of having had pains in her joints for the past 10 years. She complained of feeling tired and of becoming easily fatigued. The pain occurred at intervals and, at one time or another, involved almost every joint in her body. She dreaded the cold because she felt worse during cold weather.

Examination at that time showed a normally built white female, 63 inches in height, 34 years of age, weighing 108 pounds. Her blood pressure was 120/80 and her pulse 78 per minute. Her nasal septum was deviated to the right, both tonsils were enlarged and diseased. The thyroid gland was palpable but not enlarged. There was no evidence of deformity of any of the joints. The skin was dry, coarse, and scaly. The abdomen was tympanitic. Fluoroscopic examination revealed the fact that the entire colon was distended with flatus and could be identified without difficulty. The

remainder of the examination failed to uncover anything of pathologic significance. The serology and urine examinations were negative. X-ray of the chest was negative. The basal metabolic rate was minus 20.

The patient was started on thyroid extract in gradually increasing amounts until she received a total maintenance dose of five grains daily. The pain in the joints, while much improved, persisted; and it was felt that a tonsillectomy was indicated to remove an evident focus of infection. Urinalysis prior to the tonsillectomy was again negative for sugar.

There was a marked improvement in her general condition following the tonsillectomy. She did not complain of feeling tired, was able to read without falling asleep and could sit in a chair for several hours without feeling stiff when she attempted to rise. The thyroid extract was increased to eight grains daily. Her weight remained constant. The pulse rate was under 100 per minute and the blood pressure remained unchanged. An electrocardiogram revealed a right axis deviation.

In the spring of 1938, the patient again began to complain of pains in her joints. A urine examination was again negative for sugar and albumin. The pains persisted, and on August 21, 1938, the patient was admitted to the hospital for complete investigation. The results of the studies were as follows: hemoglobin—67 per cent, red blood cells—3.85 m., white blood cells—13,300, polys—52 per cent, lymphs—44 per cent, monos—4 per cent, sedimentation rate—25 mm. in one hour. The blood chemistry values expressed in mgm. per 100 cc. of serum revealed the following: Sugar—135 mgm., urea—23.6 mgm., uric acid—4.2 mgm. The basal metabolic rate was plus one. Urinalysis revealed 0.2 per cent of sugar on one of five occasions. The icteric index was 10.7.

X-ray of the skull revealed a normal sella turcica. An incidental finding was the presence of calcification anterior to the sella turcica in the fronto-temporal region suggesting the possibility of the presence of an osteoma.

The cervical spines were radiographically normal. Roentgenograms of the joints of the knees, elbows, wrists, and hands failed to reveal evidence of pathology of the osseous structures.

A glucose tolerance test was advised and refused. On discharge a diagnosis of atrophic rheumatoid arthritis, secondary anemia, and dysendocrinism was made.

Comment

There is a large experimental literature which deals with psychological factors in the etiology of diabetes. Allen[6] called attention to the fact that latent diabetes might be made active by psychic shock; and he, therefore, felt that such a shock might at least be an exciting cause of diabetes. Cammidge and Howard[7] were of the opinion that, in a small proportion of cases, transitory hyperglycemia and glycosuria could be induced and that the progress of an already existing diabetes might very possibly be influenced by emotional disturbances. Twenty-two cases of mental disorders associated with diabetes were reported by Menninger[8] together with a review of the experimental and clinical literature. Menninger divided his cases into three groups: one group in which the diabetes and the mental disorder apparently developed together; another group in which the diabetes developed in the course of a mental disorder; and a third group in which the diabetic condition was known to have existed before the onset of mental symptoms.

In an attempt to ascertain the effects of metrazol on the various metabolic processes, Low[9] and his associates studied the pH, CO_2, dextrose, and calcium levels of samples of blood drawn from 10 patients before and after the paroxysms induced with metrazol. These investigators found that there was an elevation in the blood sugar which they considered to be hardly significant.

The chemical changes occurring in the blood of 58 patients with psychogenic disturbances treated by metrazol convulsions were studied by Maurer[10] and his coworkers. They noted that the blood sugar levels just prior to the injection of metrazol varied from 134 to 140 mgm. per cent. At the termination of the clonic convulsions, the blood sugar levels reached values above the kidney threshold. The highest blood sugar level noted in this group of cases was 206 mgm. per cent. The rapid conversion of lactic acid

to glucose during the stage of clonic convulsions, with the resulting heaping up of the blood sugar, is offered by these authors as an explanation for this observation. No mention was made of follow-up studies of the blood sugar levels subsequent to the course of therapy.

Loman[11] and his associates drew arterial blood, as well as venous blood from the internal jugular vein of their patients, and estimated the oxygen and sugar levels and the carbon dioxide combining power. They found that the level of the blood sugar was regularly elevated. Its level gradually increased as the convulsion progressed and reached its maximum several minutes after the seizures. The curve illustrating the rise in the sugar level of the arterial blood closely paralleled that of the venous blood. This work confirmed the observations made by Maurer and his coworkers.

A single case report hardly calls for a prolonged discussion. But the observation recorded was sufficiently perplexing to the author to warrant this presentation. The blood sugar level noted during the first hospital admission and the presence of a small amount of sugar in the urine on one occasion do not constitute enough evidence for a diagnosis of diabetes mellitus, in the absence of the subjective evidences of this metabolic disturbance. One cannot be sure that this case was one of latent diabetes, in view of the fact that many subsequent urinalyses over a period of three years failed to show the presence of sugar. One can only speculate concerning the relationship of the metrazol therapy to the diabetic picture which resulted following the short series of convulsive seizures induced in this patient. The mechanism of this disturbed sugar metabolism remains entirely unknown.

Summary

A case of diabetes mellitus which developed during the course of metrazol shock therapy is reported.

85 Eastern Parkway
Brooklyn, N. Y.

REFERENCES

1. von Meduna, L.: Versuche über die biologische Beemflussing des ablaufes der Schizophrenic, Campher-und cardiazolkrampfe, Ztschr. f. d. ges. Neurol. u. Psychiat. 152:235, 1935.
2. von Meduna, L., and Friedman, E.: The convulsive therapy of the psychoses. J. A. M. A. 112:6:501, February, 1939.
3. Hutchings, R. H.: Personal communication to the author.
4. Epstein, J.: Aplastic anemia arising during metrazol treatment of schizophrenia: Report of a fatal case. PSYCHIAT. QUART. 13:3:419-428, July, 1939.
5. Wender, L., and Epstein, M. D.: Disturbances in the reticulo-endothelial system during the course of metrazol treatment. PSYCHIAT. QUART. 13:3:534-538, July, 1939.
6. Allen, F. M.: Studies concerning Glycosuria and Diabetes. Wm. Leonard, Boston, 1913, pp. 755-757.
7. Cammidge, J. P., and Howard, H. A. H.: New Views on Diabetes Mellitus. Henry Frowde, London, 1923, pp. 165-169.
8. Menninger, Wm.: Psychological factors in the etiology of diabetes. J. Nerv. and Mental Diseases. 81:1:1-13, January, 1934.
9. Low, A. A.; Sonenthal, I. R.; Blaurock, M. R.; Kaplan, M., and Sherman, I.: Metrazol shock treatment of the functional psychoses. Arch. Neurol. and Psychiat. 39:717-736. April, 1938.
10. Maurer, S.; Wiles, H. O.; Marbery, C. M.; Skorodin, B., and Fisher, M. L.: Blood chemical changes occurring in the treatment of psychogenic mental disorders by metrazol convulsions. Am. J. Psychiat. 94:17:1355-1362. (May), 1938.
11. Loman, J.; Rinkel, M., and Meyerson, A.: Metrazol convulsions: changes in oxygen, carbon dioxide, and sugar content of arterial and internal jugular venous blood. Arch. Neurol. and Psychiat. 43:682-692. (April), 1940.

PSYCHOSIS WITH HUNTINGTON'S CHOREA*

BY DAVID ROSENBAUM, M. D.

Huntington's chorea is a disease entity which was apparently well known to the laity for many generations before an adequate description was recorded in medical literature. George Huntington wrote his classical description of this hereditary form of chorea[1] in 1872. It is true that previously, several physicians had reported the existence of the disease—Thilenius[2] in 1816 was probably the earliest—but Huntington's was the most complete of the earlier accounts and the first to receive international attention.

For centuries, hereditary chorea has been known as "magrums" or "megrims" to members of the laity familiar with the condition. (This name was given to it by the early Dutch settlers.) In colonial New England, a number of persons afflicted with "magrums" were burned as witches[3] and it was popularly believed that the families suffering from it were cursed because an ancestor had dared to pantomine derisively the sufferings of Christ on the cross. Choreatics were observed by Huntington when he was a small child riding in his father's buggy; he later said that he was so impressed by the sight of these people that he never forgot them.

Huntington stressed three features: (1) the hereditary nature; (2) the onset in middle life; and (3) the progressive dementia.

The first of these features, the hereditary nature, is now well known and requires little discussion here. It is apparently a dominant Mendelian character and any parent with chorea may expect to transmit the disease to half his children. If the required gene is present in the chromosome complement of any person, the manifestations of the disease will appear, usually in middle life, unless death intervenes. Another point worth repeating here is that aptly made by Huntington, that "it never skips a generation to manifest itself in another, once having yielded its claims, it never regains them."

The fact that the disease usually first reveals itself in middle life is also well recognized, but it is not well known that many instances have been described of onset at other periods in life. Stone and

*Read before the interhospital conference, held at the Utica State Hospital, Utica, N. Y., April 26, 1940.

Falstein[4] reported a series of cases in which the onset occurred in all decades up to the ninth, although in their group, as in most others, the predominating age of onset was in the fifth decade.

The last of the three features stressed by Huntington, the progressive dementia, is the element of the disease which concerns us most. It was considered of interest to collect a group of cases from the records of the St. Lawrence State Hospital and to analyze them, chiefly from the point of view of descriptive psychiatry.

A study of the admissions to this hospital over the past 25 years shows 46 instances of Huntington's chorea, of which 27 occurred in males and 19 in females. All of them, as far as can be determined from the existing records, are authentic.

The family history in 40 of the 46 cases showed the presence of the disease in other members of the family. In the six cases in which the family history was said to be negative, the information was so incomplete as to make it impossible to state with any degree of certainty that there had been no other instances of the disease in the family. Many of the patients were related to one another, and most of them showed a large number of afflicted relatives in their genealogical trees.

The average age of onset of the choreiform movements in this group was 45 years, with the earliest at 15 and the latest at 61. The average age of the onset of the psychosis was 46, with the earliest onset given as 19 and the latest as 64. In many cases it was impossible to date definitely the onset of the psychosis, as many of the patients were apparently mentally deficient, and others had always been considered "eccentric." It has been stated that the members of the families afflicted with this hereditary blight are constitutionally inferior, but although this was apparently true of most of the present group, some patients who exhibited classical pictures of the disease had been considered intellectually superior.

Dynan[5] stated over 25 years ago that about one-third of his patients exhibited mental symptoms first and that two-thirds exhibited the purely neurological manifestations first. Analysis of the present group showed that 14 patients exhibited chorea before the onset of psychotic symptoms; eight patients developed their psychosis as the initial manifestation, and 14 patients experienced an apparently simultaneous onset of both chorea and psychosis. It

has been noted by some observers that certain families show a tendency for the chorea to occur first, while in other families the opposite tendency is noted. The family histories in the cases here presented, on the whole, were not sufficiently detailed to permit adequate analysis of this point.

The outcome of the disease in the present group of cases clearly supports the universally-held idea of the prognosis. It is invariably stated that the prognosis is hopeless. The following tabulation indicates the manner of termination in the present series:

Died in the hospital	27
Still in the hospital	11
Self-destruction	2
Discharged	4
Paroled	2

The average age at the time of death was 54 years, and the average age of the patients at the time of discharge from the hospital and of those still alive in the hospital was 57.

The pathology of Huntington's chorea has been described in great detail by a number of authors. Probably the best description is to be found in Dunlap's[6] monograph. Stone and Falstein,[7] in their recent study, state that the disease is essentially an atrophic process involving the brain as a whole, associated with bilateral lesions in the neostriatum and in the cerebral hemispheres, with a definite loss of parenchymal cells in the involved regions. They believe the fundamental nature of the change to be a premature atrophy of the cells of the cerebral cortex and the neostriatum.

Study of the symptoms exhibited by these patients reveals several interesting facts. The most frequent psychiatric symptom seen in this group was emotional instability, generally manifested as irritability. Only five of the entire group of 46 did not show irritability. The degree varied within wide limits in different patients, but in a surprisingly large number it was one of the most outstanding symptoms and frequently the one which led to hospitalization. Five patients became progressively irritable to the point of attempted homicide. In none was the homicidal attempt felt to be due to paranoid ideas—rather was it attributed to explosive irritability. Thirty patients were assaultive during their hospitalization. In a few instances the assaults were believed to

have been impulsive, particularly in badly deteriorated patients, but more often they were in irritable patients who had impaired judgment.

The next most frequent psychiatric manifestation was frank mental deterioration. This was exhibited by 35 of the 46 patients; of the minority of 11 who failed to show it, 10 were either paroled or discharged or were relatively early cases still in the hospital. Of the 46 patients in the entire series, 31 showed defects in orientation and 42 showed defects in memory, mental tension or attention. The mental deterioration in most patients who were followed long enough progressed to a typical amentia: They began to wet and soil; they had to be spoonfed; their habits were untidy; and eventually they became entirely inaccessible.

Delusions of various types were present in 38 of the patients, the most common type by far being paranoid ideas, which were entertained by 32 patients. These were usually ideas of persecution directed against members of their families, friends, etc., and were rarely systematized, even early in the disease before any deterioration had appeared. In 13 instances, the persecutory ideas were associated with or replaced by grandiose delusions.

Hallucinations were present in 27 patients, and possibly others also had been subject to hallucinatory experiences before coming under medical observation. The hallucinations showed great variety in type and content, and no generalizations concerning them seem advisable. Some of the earlier descriptions of the psychosis accompanying Huntington's chorea, such as Hamilton's,[8] in 1908, state that hallucinations are uncommon. Some of the textbooks make the same statement, apparently basing their assertions on these sources. It is interesting that more than half the patients in this series were hallucinated.

Another point worthy of investigation is the incidence of convulsions. In 1931, Notkin[9] reported an instance of Huntington's chorea with definite convulsions. In the same paper he reported the result of a survey of the literature for similar cases. He succeeded in finding 21 others, but careful examination of the cases as he describes them shows that many of them are questionable. He apparently believes that there may be some relationship between Huntington's chorea and the idiopathic group of convulsive

states. In the group of patients under discussion, none suffered from convulsions and as far as could be determined only one showed the presence of epilepsy in the family history. It would seem safe to say that no relation could be found in this series between hereditary chorea and epilepsy.

A feature of the disease which is mentioned frequently is the tendency to depression and self-destruction. Depression was noted in 13 patients and two of these ended their lives by suicide. Furthermore, frequent mention is made in the family histories of suicides by afflicted relatives of the patients. The depression is noted rather early in the course of the disease; both of these suicides occurred before the patients had become deteriorated and while at least partial insight was present. It is easy to understand why depression and its effects should be fairly common in people who suffer from an affliction the hopelessness of which they know only too well, as most of them have observed the entire course of the disease in members of their immediate families. This is true particularly among the more intelligent and educated choreatics who have a more complete realization of the gravity of the situation.

The following are brief summaries of a few illustrative cases.

Case Material

Case 1. This patient was a 29-year-old white male, admitted to the hospital on September 14, 1927. It was said at that time that he had never had a permanent home and that he was accustomed to wander about from place to place. He had shown the first signs of chorea 10 years previously and they had been progressive. His mother had had Huntington's chorea, and died in a psychiatric hospital. His maternal grandfather, great-grandfather and aunt had all suffered from the same disorder. He had served several terms in prison for vagrancy. The informant, with whom he had been living sporadically, said that the patient would get up at night as if suffering from hallucinations, turn on the lights and expose himself before members of the family. On admission, he expressed himself relevantly and coherently, and although he said he was depressed he frequently laughed. He was correctly oriented and showed no organic defects. His insight was good. During his hospitalization, he became gradually deteriorated. Three years after admission he had become silly, and lost his insight. Two years later he developed paranoid delusions, expressing ideas of poisoning. At about the same time, he showed irritability and began to be hallucinated.

By 1934, seven years after admission, he had become inaccessible and untidy, and a speech defect had developed to such an extent that his mumbling was unintelligible. He developed a lung abscess and died at the age of 36. This is a typical case, except that the choreiform movements and the psychosis had their onset at a much earlier age than usual. The disease progressed at about the same rate as it does after a later onset.

Case 2. This 47-year-old physician was admitted to the hospital on October 14, 1928. His paternal grandmother had had Huntington's chorea, and his father had died at the age of 25 of tuberculosis. The patient was graduated from high school at 16, taught in a country school for two years, and then studied medicine, obtaining his degree at the age of 23. While a medical student, he apparently brooded a good deal about the presence of Huntington's chorea in his family, and he became addicted to morphine. Following his internship, he entered private practice and never remained more than two or three years in any one place. At various times he was addicted to morphine, and throughout his adult life he was a "heavy drinker." He never married. At the age of 44, he told a colleague that he was developing chorea and at about the same time he began to exhibit irritability. This symptom led to his hospitalization. On admission, he showed marked choreiform movements, defects in attention and immediate recall, could not do the serial sevens, but showed good insight. He remained in the hospital only 19 days and then insisted on leaving. Attempts at followup were unsuccessful and his subsequent course is not known. This case illustrates the effect on an intelligent person of the presence of Huntington's chorea in the lineage. During the greater part of his life he brooded about the possibility of developing hereditary chorea, and as a result became an alcoholic and a morphine addict. His instability and his inability to continue his practice of medicine in any one place for any long period might also have been due to his anxiety.

Case 3. This patient, a 41-year-old white male, was admitted to the hospital on July 4, 1907. His mother, six maternal aunts, five maternal uncles, a brother and a sister had Huntington's chorea, and one of his afflicted aunts had ended her own life. The patient had never attended school and had been a wandering vagrant for many years. At the age of 38 he began to exhibit choreiform movements and at the age of 40 he was noted to be psychotic. He would become very excitable and irritable, he threatened to kill people with an axe, and at times he expressed the wish to die. At other times he was incoherent. On admission he showed marked choreiform movements, a speech defect and evidences of impairment of mental functions. He was partially disoriented, showed memory and mental ten-

sion defects, and lack of insight. He was irritable while in the hospital and although he worked in the hospital industrial shop for a short time he soon began to deteriorate progressively. Within three years, a speech defect developed, so marked that his production could no longer be understood. He was unable to dress himself or to walk without assistance, and had difficulty in swallowing. His course was rapidly progressive and he died less than five years after admission. For some time before his death, he was in a gradually developing and deepening coma. At autopsy, only atrophic changes in the brain were found; hence this appears to have been a death due to Huntington's chorea *per se*.

In the final state of amentia, these patients, were it not for their grotesque choreiform or athetotic movements and their facial grimacing and contortions, would be indistinguishable from badly deteriorated schizophrenic or paretic patients. However, in a member of an afflicted family, when one has the opportunity to see the irritability, the assaultiveness, the depression, the hallucinations, the paranoid ideas and the beginning evidences of organic defects, it should be possible to make the diagnosis of "Psychosis with Huntington's Chorea" even before choreiform movements are noted.

St. Lawrence State Hospital
Ogdensburg, N. Y.

REFERENCES

1. Huntington, G.: On chorea. Med. and Surg. Reporter (Philadelphia) 26, 317, April, 1872.
2. Thilenius: Mentioned in 4.
3. Vessie, P. R.: On the transmission of Huntington's chorea for 300 years—the Bures family group. Jour. of Nerv. and Ment. Dis., 76:6, 553-573, December, 1932.
4. Stone, T. T., and Falstein, E. I.: Geneological studies in Huntington's chorea. Jour. of Nerv. and Ment. Dis., 89:6, 795-809, June, 1939.
5. Dynan, N. J.: The physical and mental states in chronic chorea. Am. Jour. of Insanity, 70:3, 589-636, January, 1914.
6. Dunlap, C. B.: Pathologic changes in Huntington's chorea. Arch. of Neur. and Psych., 18:6, 867-943, December, 1927.
7. Stone, T. T., and Falstein, E. I.: Pathology of Huntington's chorea. Jour. of Nerv. and Ment. Dis., 88:5, 602-626, November, 1938; 88:6, 773-797, December, 1938.
8. Hamilton, A. S.: A report of twenty-seven cases of chronic progressive chorea. Am. Jour. of Insan., 64:3, 403-475, January, 1908.
9. Notkin, J.: Convulsive manifestations in Huntington's chorea. Jour. of Nerv. and Ment. Dis., 74:2, 149-160, August, 1931.

SCHIZOPHRENIC-LIKE REACTIONS IN CHILDREN*

Preliminary Report: Studies by Electroencephalography, Pneumoencephalography and Psychological Tests

BY E. R. CLARDY, M. D., L. N. GOLDENSOHN, M. D., AND KATE LEVINE, M. A.

Schizophrenia in children has long been a controversial subject. Some investigators believe that it does not occur under 12 years of age. MacKinnon[1] believes that true schizophrenia rarely occurs in children under the age of 10, and then not unless puberty has been reached. In some cases of so-called schizophrenic children, evidences of organic brain disease have been demonstrated by pneumoencephalography. More recently, electroencephalography has afforded another means of study.

The writers have attempted an investigation of schizophrenia, or schizophrenic-like reactions, in children. This preliminary report is the result of research to determine the possibility of the presence of organic factors in these children.

In a series of seven cases, the following techniques have been used: (1) pneumoencephalography, (2) electroencephalography, and (3) psychological tests (Revised Standford-Binet, Form L; Weigl Object Sorting and Color Form Sorting Tests, Vigotsky blocks, Kohs block designs and the Rorschach method). Each patient was studied psychiatrically for a period of three to seven years at the Rockland State Hospital (children's group) and allied hospitals.

Case Material

Case 1. *History:* R. S., a 10-year-old white boy of German parentage, was admitted to the New York State Psychiatric Institute on May 15, 1933. The history stated that his conduct had been "very peculiar." He suffered from insomnia, was afraid of the dark, would pull the bed-clothing over his head, had a hand-washing mania and showed an aversion to anything which was not spotlessly clean. The main problem, according to the mother, was that he made strange buzzing sounds which almost "drove her frantic." He would stand out in the yard and buzz like an automobile for hours at a time. He did not play with other children but preferred to remain buzzing out on the lawn.

*Read before the interhospital conference held at the New York State Psychiatric Institute and Hospital, New York., N. Y., April 18, 1940.

In the family history, it was noted that a paternal aunt had experienced a "nervous breakdown" and was confined to a mental hospital. The mother was a stable, dependable type of woman. The father was a heavy drinker who died of delirium tremens when the patient was about seven years old. At the time of his father's death, the boy developed facial tics.

The boy's birth and early development were described as normal. When only three or four years of age he was afraid of animals, especially chickens. Sad music and sad pictures upset him greatly and occasionally caused him to cry. In school he was somewhat dull and was rather poor in arithmetic. There was no history of diseases or serious injuries. When he was six years old, however, he was hit on the head with a stone. The injury was slight, but subsequently he was afraid of children and refused to play with them.

Course: During his residence at the Psychiatric Institute, from May 15, 1933 to January 3, 1934, he was often preoccupied with fantasy and at times appeared to be somewhat confused. He was discharged with the diagnosis of dementia præcox, hebephrenic type. A few weeks later he was admitted to the Kings Park State Hospital. During his residence there, no evidence of delusions or hallucinations was found, but he was seclusive and reluctant to play with other boys. He also remained afraid of chickens.

Following his transfer to the Rockland State Hospital on February 11, 1937, he was seclusive and fearful. Sometimes he would stand in the corner of a room, rock back and forth, and rub his arms against the wall. Other manneristic actions were noted; for instance, he would walk down the hall with his eyes shut and his arms extended. One characteristic was extremely interesting, that is he seemed to have an unusual ability to remember dates. He could remember hundreds of events of minor importance—the exact hour, day, month, and year of their occurrence. The boy's actions and condition apparently have not changed over a period of seven years.

The physical examination was negative. Laboratory studies revealed no abnormalities. Careful neurological examination revealed no abnormalities. A pneumoencephalogram showed some hypoplasia of the right cerebral hemisphere and some consequent

enlargement of the lateral ventricle on that side. In the electroencephalogram the only thing of note was slow waves, less than seven a second, particularly in the occipital region.

It was noted that before the boy came to the hospital, on psychometric examinations (Stanford-Binet Tests), an I. Q. of 77 was given to him in 1933 and 76 in 1934. The revised Stanford-Binet administered on June 1, 1939, gave him a mental age of 8-0, and an I. Q. of 53, suggesting true intellectual deterioration. In sorting tests and in the Kohs block designs, he had difficulty in grasping and applying abstract conceptual principles. Interpretation of the Rorschach record indicated that the range of content and the breadth of ideas expressed were out of all proportion to the poor quality of perception and the constricted range of responsiveness. The general impression from the Rorschach record was that the patient was an intellectually deteriorated schizophrenic, who had an impaired capacity for adequate and adjusted responsiveness to life, rather than any severe disorganization or emotional disturbance.

Comment: In this case we have a boy with some psychoneurotic symptoms. The pneumoencephalogram reveals the presence of definite though minimal organic involvement of the brain. The electroencephalogram showed only slow waves, which have often been found in schizophrenic as well as in other conditions. The principal mental symptoms have been those of schizophrenia: withdrawal from reality, bizarre ideas and actions, plus evidence of deterioration.

Case 2. *History:* L. N., a nine-year-old white boy of mixed race, had a history of maladjustment since the age of about four years when it became apparent that he did not play normally with other children, was overseclusive, and reacted unfavorably to discipline. Finally he was removed from school because of inattention, silly behavior, extreme fantasy, daydreaming and infantile reactions.

The family history indicated that the father was an ineffectual, irresponsible, narcissistic individual. The mother was an unstable person who often took flight in alcoholic bouts. During these episodes, she had suicidal ideas. She admitted that she probably had rejected the patient, holding up his seven-year-old brother as a shining example.

Course: Following admission to the Rockland State Hospital Children's Group, on January 17, 1937, the child continued to be seclusive, silly and superficial. His actions were usually rather bizarre. At one time he tried to plant the blossom of a flower in a pile of rocks. There was also evidence of eidetic imagery, in that he could project visions at will upon the walls and ceilings. These visions had some actual basis concerning stories he had read or pictures he had seen. There were a number of psychoneurotic manifestations, such as ties and facial spasms. At one time he expressed a compulsive idea urging him to kill a boy. During a residence of three years at the hospital, the child showed little change. He remained superficial, silly and extremely introverted.

On physical examination, he was found to be obese with an effeminate body contour. The genitals were small, and the right testicle was undescended. The general body type simulated a Fröhlich syndrome. Neurological examination gave these positive findings: deep reflexes hyperactive, with an increased patellar reflex on the left; exhaustible abdominal reflexes; slight facial weakness, more marked on the left; anisocoria with the larger pupil on the left; defective plantar flexion bilaterally with dorsiflexion of the big toe on the left. A pneumoencephalogram obtained on October 25, 1939 showed the lateral ventricles to be slightly enlarged, the right being larger than the left. The impression was that of mild bilateral cerebral hypoplasia. In May, 1939, an electroencephalogram revealed nonequivalence on the two sides and slow waves of less than seven a second.

The psychometric examinations were as follows: Stanford-Binet in 1937: mental age 13-2, I. Q. 134. A revised Stanford-Binet test on November 15, 1939, gave him a mental age of 13-10 and I. Q. of 109. A Rorschach record obtained in December, 1939, did not reveal the signs usually associated with organic brain disease in the adult patient. The basic personality configuration was one in which the increased withdrawal from objective reality into fantasy, coupled with the impulsive emotional responses to reality, might provide a ready setting for a schizophrenic reaction. The manner of perception, with ready elaboration, was more nearly like that of the schizophrenic than like that of the patient with organic brain disease. There was no evidence of impairment of abstract ability

in sorting tests or in performance with the Kohs block designs. Although there has apparently been some intellectual deterioration, as indicated by a drop of 25 points in the intelligence quotient, he is still able to apply abstract conceptual principles.

Comment: In this case an introverted boy, nine years of age, developed a frank psychosis. From the standpoint of heredity, the predisposing factors were: an unstable, alcoholic mother and an irresponsible, ineffectual, narcissistic father; rejection of the boy by his mother; sibling rivalry. There was evidence of dysendocrinism and organic brain affection. The presence of the latter was demonstrated by (1) the neurological findings, (2) the electroencephalogram, which showed slow alpha waves and nonequivalence of the two cerebral hemispheres, and (3) the pneumoencephalogram, which showed the lateral ventricles to be unequal and enlarged. The final general interpretation of the picture was mild bilateral cerebral hypoplasia. The actual mental picture resembled schizophrenia, although there were some psychoneurotic symptoms.

Case 3. *History:* J. B., a Negro boy, became maladjusted when he began school at about six or seven years of age. At that time, he daydreamed, had no regard for discipline, and would run about the room disorganizing the class. He was sensitive, shy, seclusive, and fought with other children. At one time his aunt found him confused and irrational. He also refused to eat and said that people were whispering about him.

The family history was negative. The patient's father died of lobar pneumonia at the age of 32. The mother was a moody, unstable, irresponsible person, who never admitted that she was the child's real mother. The patient was the only child.

His birth and early development were normal. He suffered no serious illnesses. At the age of five he fell out of a tall tree, but seemed unharmed and was not unconscious. He said that at the age of 13, he had fallen from a horse and injured his head severely. Records from the school which he attended at that time stated that there had been no such accident. He was admitted to Bellevue Hospital on June 27, 1936. He was described as depressed, restless, resistive, assaultive and delusional. He accused himself of

having done "something wrong" and asserted that someone was going to kill him. He also refused to eat because he feared that the food was poisoned.

Course: Following admission to the Rockland State Hospital Children's Group, on July 3, 1936, he said he had heard voices telling him to hit his head against the wall; and, consequently, he thought the voices were coming from people who wished to kill him. He was well oriented and had a good memory for more recent events, but his memory was fragmentary concerning the period just prior to going to Bellevue. About every three or four months he would become either excited or depressed; and afterward delusions and hallucinations would develop. He said that he heard voices telling him that he was an illegitimate child. He also feared that his stomach and chest would "fall out" or that something would happen to his brain. He believed that other children wanted to poison him because he was a "bad boy." The phrases of depression, excitement, delusions and hallucinations would last from one to two months, following which he would usually have a complete remission.

On September 4, 1938, during one of these periods of remission, he was paroled and subsequently discharged. The diagnosis at that time was dementia præcox, mixed type. He did well at home for about five months, then became sulky, stubborn, and talked to himself. Consequently, he was returned to the children's group on March 10, 1939.

The general physical examination was negative except for a pigeon breast. Laboratory studies revealed no abnormalities. A roentgenogram of the skull showed no evidence of fracture. Repeated detailed neurological examinations revealed no abnormalities. A pneumoencephalogram obtained April 5, 1939, showed evidence of some dilatation of the left lateral ventricle, presumably caused by hypoplasia of the left cerebral hemisphere. An electroencephalogram made in May, 1939, revealed slow alpha waves, less than seven a second, particularly in the occipital region.

Psychological examinations were as follows: In August, 1936, the I. Q. was 85; in May, 1939, a Revised Standford-Binet, Form L, gave an I. Q. of 82. The patient had no difficulty with the Kohs blocks. In sorting tests, there was difficulty in the application of

abstract concepts, although the boy's performance was better after demonstration. An interpretation of the Rorschach record on May 23, 1939, stated: Inner activity was almost entirely wiped out and there was complete inability to respond to environmental stimuli. The record suggested organic involvement of the brain, in that there were only 12 responses, no movement responses, only one use of popular concept, low form plus per cent, repetition, perplexity, and expression of impotence. The manner of perception was also characteristic of the organic case.

Comment: This case was similar in many respects to the preceding one. The psychiatric picture was frankly schizophrenic, except for some periods which, in retrospect, suggested organic confusion. The mental symptoms began with maladjustment when the patient was six or seven. The pneumoencephalogram revealed definite evidence of organic brain affection. Electroencephalography indicated the presence of slow waves. The psychological tests failed to reveal any evidence of organic disease of the brain, but the Rorschach examination indicated the probability of an organic reaction.

Case 4. *History:* On April 5, 1934, L. M., an Italian boy of 10 years, was sent to the Bellevue Psychiatric Hospital. The problem presented was one of asocial behavior, assaultiveness, tantrums and extreme negativism. His difficulties began at the age of seven, when the school authorities described his behavior as "very peculiar." For example, when he was corrected, he would put away his paper and pencil, lay his head upon the desk and make all sorts of noises. At play he preferred to be by himself, but when with other children, he continually bullied and fought with them. He frequently soiled himself.

The father was described as a chronic alcoholic. The patient's mother was troublesome, quarrelsome and unfaithful to her husband. She was reported to have beaten the patient over the head with a broom when he disobeyed or annoyed her. Of the boy's four siblings, only a younger sister presented a behavior problem. She was described as uncommunicative, restless, annoying and quarrelsome.

There was nothing unusual about the patient's early developmental history. The birth was somewhat difficult but not abnor-

mal, and he developed into a strong healthy infant. There was no history of serious illness or injury.

Course: Following the boy's admission to Bellevue Hospital in 1934, he was impulsive and assaultive toward other children. His reactions were described as of a primitive type. He was shy, manneristic and emotionally flat. He was transferred to the Rockland State Hospital Children's Group on June 15, 1934, and immediately following admission was resistive, assaultive and almost mute. When involved in difficulties with other patients, he would spit, scream, bite and scratch. Occasionally, during his mute spells, he assumed catatonic-like positions, drawing himself into a ball with his head buried in his arms and his knees up. Although the boy was preoccupied and self-absorbed, there was no evidence of delusions or hallucinations. During his residence here he showed almost no change.

The physical examination revealed a hypopituitary-like habitus. The boy's facial expression suggested *Schnauz-krampf*. The basal metabolic rate could not be obtained because of overactivity. Routine laboratory procedures revealed no abnormalities. Repeated detailed neurological examinations revealed no abnormalities. In April, 1939, pneumoencephalography disclosed a normal ventricular system. In May, 1939, electroencephalography showed slight nonequivalence of both sides of the head and slow waves of less than seven a second.

This patient was so preoccupied, withdrawn and disorganized that psychological testing was impossible. On September 29, 1939, a Rorschach test was administered, but a record sufficiently complete for satisfactory interpretation could not be obtained. On one occasion, he refused all 10 cards, with one descriptive remark about color. They were shown to him again, and he gave three responses. One of these was a popular response to a readily perceived whole concept, another was a rather good human form, and the third was a bizarre confabulation, characteristic of the schizophrenic.

Comment: This boy had presented a severe behavior problem since the age of seven. When first observed at this hospital at 10 years of age, he presented a typical schizophrenic picture. Neurologic and pneumoencephalographic examinations revealed no evi-

dence of neural involvement. Electroencephalography revealed nonequivalence of the two sides and slow waves. The Rorschach examination gave findings characteristic of schizophrenia.

Case 5. *History:* J. R., a 12-year-old boy of Irish stock, became disturbed following an attack of rheumatic fever. He would shout and scream without provocation, became extremely seclusive, would not play with other children, and was afraid of people, dogs and cats. Finally he became suspicious of his parents, believing that they talked about him.

There was nothing of interest in the remote family history. The father was described as a small, shy, timid, apprehensive individual dominated by his wife. The mother was pictured as an enormous, towering, aggressive person who was anxious about the patient's health and overprotected him. He was the fifth of six children; two died in infancy, and the others were described as normal. The patient's birth and early development were normal, but after the first year he became a "sickly" child. He developed whooping cough at 16 months, pneumonia at 20 months, measles at three years, German measles at eight years, nephritis at nine years and bronchitis every winter. There was evidence also of rickets in early childhood. In May, 1934, he developed the rheumatic fever already referred to. There was no history of serious injuries. When he began school, he was extremely retiring, shy and seclusive, and played with boys younger than himself.

Course: On July 1, 1934, he was sent to the Bellevue Psychiatric Hospital. While there, he was apprehensive, resistive, self-absorbed, unable to care for himself, and had spells of excitement. On August 22, 1934, he was admitted to the Kings Park State Hospital where he was diagnosed dementia præcox, simple type. He was transferred to the Rockland State Hospital Children's Group on August 25, 1936, where he remained bewildered, confused, listless, apathetic, and out of contact; however, there was no evidence of delusions or hallucinations.

General physical examination revealed a thin child whose tonsils were large and infected. Roentgenographic examination showed the heart to be slightly enlarged. Routine laboratory studies revealed no abnormalities. Detailed neurological examination showed no abnormalities. A pneumoencephalogram made on June

7, 1939, was interpreted as showing internal and external hydrocephalus and cortical atrophy in the parietal region. The electroencephalogram obtained in May, 1939, revealed slow alpha waves of less than seven a second and nonequivalence of the two sides.

Psychometric examinations were as follows: 1934 Stanford-Binet Test (Bellevue Hospital) M. A. 11-8, I. Q., 96; September 13, 1936, Stanford-Binet (children's group) M. A. 10-9, I. Q. 76; May, 1939, Revised Stanford-Binet, M. A. 12-0, I. Q. 80. The entire performance on the latter examination was characterized by poor contact with the situation, withdrawal and preoccupation.

Performance tests, September 25, 1939, were interpreted as follows: The Weigl forms were handled in a normally intelligent manner. With the Vigotsky blocks, the patient was unable to grasp the basic scheme, even after demonstration, and could not repeat the performance. With the Kohs block designs, he experienced great difficulty; the impression was that the difficulty in analysis might be related to his general inability to enter fully into a concrete situation. The Rorschach interpretation, September 25, 1939, was that the brief record pictured a high degree of constriction, i. e., withdrawal from stimulating situations. There was a complete evasion of any responsiveness to emotional stimuli from without, but inner activity approached a mature level. There were no bizarre or disorganized responses. The record pictured a person incapable of responding adequately to his environment. The somewhat limited perceptions of this record and the lack of elaboration or careful location are ambiguous in interpretive value. They may represent the characteristic manner of perception of the organic patient, or they may represent the patient's poor contact and withdrawal from the examination situation.

Comment: This case is that of an overprotected boy who had an extremely introverted personality. The onset of his psychosis occurred following an attack of rheumatic fever. The mental and behavior pictures were chiefly those of schizophrenia. The psychological examinations indicated withdrawal and inability to respond to environmental factors, but no true deterioration or disorganization. The neurological examination revealed no evidence of organic disease of the central nervous system. Pneumoencephalography revealed cortical atrophy and moderate internal and exter-

nal hydrocephalus. Electroencephalography revealed the presence of slow waves and nonequivalence on the two sides of the head.

Case 6. *History:* At the age of 11, L. K., a Jewish boy, became exceedingly quiet, would answer questions only after they were repeated several times, and assumed bizarre attitudes such as standing in the corner of a room for long periods. He frequently set fires.

The family history was not abnormal. A younger brother was described as normal. The father was a meek, mild-mannered individual dominated by his wife. However, he annoyed L. about his school work, teased and belittled him.

The boy's birth was described as normal, except that the labor lasted 27 hours. At 13 months, he underwent a mastoidectomy. He experienced a series of convulsions beginning at the age of three. Five or six seizures would occur over a period of about one year, then they would cease. Recurrences came at the ages of five and eight. In the child's early development, the mother described him as "abnormal." She said that he was seclusive, played with his own body, and sucked his thumb all night. Masturbation became excessive at the age of four and has been a continuous habit since. He was overconcerned with sexual matters, lifted little girls' skirts, and attempted to play with his mother's legs and breasts.

Course: Following admission to the Rockland State Hospital Children's Group on August 28, 1936, he was seclusive, extremely quiet, apprehensive and anxious. Masturbation was practiced openly, and he made sexual advances toward teachers, nurses and attendants. On October 18, 1937, he started a six-weeks course of hypoglycemia. During the treatments he had three epileptic seizures but no comas. Treatment was finally discontinued because there was no improvement. During the past year, he began to experience hallucinations.

Physical examination showed his body build to be somewhat effeminate. Laboratory studies revealed no abnormalities. Careful neurological examination showed no abnormalities. On pneumoencephalographic examination performed January 17, 1940, no abnormalities were detected. Electroencephalography, performed in

May, 1939, showed some nonequivalence on both sides of the head, and slow waves of less than seven to the second.

Results of psychometric examinations were: October, 1936, Revised Stanford-Binet, Form L, M. A. 13-8, I. Q. 101; December 8, 1939, M. A. 12-0, I Q. 80. There appeared to be some degree of intellectual deterioration. Performance on Kohs block designs, March 16, 1940, did not show specific impairment of capacity for abstraction, but there was a generalized inability of the patient to free himself of inner preoccupation in order to grasp more complex situations. Performance with Weigl forms and Vigotsky blocks showed interference with efficient function of the so-called abstract intellectual powers, but these were still existent and could be called into use.

The Rorschach examination demonstrated an unmistakable picture of a schizophrenic process at work in an individual originally of at least average ability; consequently bizarre and normal high level responses were found side by side; and there was a gradual withdrawal from all forms of reality. The constructive forces had not wholly destroyed the capacities of the boy for adjustment, but had distorted and disorganized them.

Comment: This boy has a history of convulsions up to the age of eight which might suggest organic brain affection; however, the pneumoencephalogram was negative. The electroencephalogram showed slow waves and some nonequivalence of the two hemispheres, but the writers do not deem this conclusive evidence of organic pathology. On the other hand, they note typical schizophrenic symptoms which probably began in early childhood. Rorschach interpretation and psychometric examinations disfavored organic brain pathology and described schizophrenic-like reactions. The patient also showed considerable evidence of deterioration.

Case 7. *History:* In the summer of 1934, W. H., a 12-year-old white boy of Jewish stock, became depressed, retarded, and found it impossible to continue in school. On October 15, 1935, he was admitted to the New York State Psychiatric Institute; during his residence there he showed marked catatonic manifestations and was actively hallucinated. He was diagnosed dementia præcox, catatonic type.

The mother was described as a "very high-strung," neurotic individual. The father died at the age of 39 of carcinoma of the lungs. He was described as tense and frequently depressed. It was immediately following his death that W.'s illness began.

Course: On May 18, 1936, W. was transferred to the Rockland State Hospital Children's Group. On mental examination, he was silly and superficial, he grimaced and made queer noises. When asked questions, his only answer was, "I don't know." He wet, soiled, and actively hallucinated. During his residence at Rockland his condition changed frequently. On occasions he was mute, assumed catatonic postures, and would not react to external stimuli such as pin-pricks. At other times, with the exception of silliness, he appeared almost normal. He occasionally attempted intercourse with small girls and made advances also to the teachers. Masturbation was frequently observed. During an observation period of two years the boy's behavior did not change noticeably, except that there were fewer of the "near normal" periods.

The physical examination was negative except that the patient had an infantile growth of hair about the genitals and other parts of the body. The genitals were hypoplastic. Routine laboratory studies were negative. Detailed neurological examination was negative. A pneumoencephalographic series revealed slight bilateral dilatation of the lateral ventricles. There was a greater dilatation on the right than on the left. There seemed to be some shortening of the left posterior horn. In May, 1939, an electroencephalogram showed a marked asynchronism of waves of the two hemispheres and slow waves of less than seven a second. Psychological examinations were: June, 1939, Revised Stanford-Binet, Form L, M. A. 15-7, I. Q. 104. There was some blocking and confusion without evidence of genuine intellectual deterioration. The patient sorted Weigl forms according to the two possible categorical principles and generalized the reasons without difficulty.

The Rorschach record was one in which the concrete approach to practical reality was ignored. Inability to cope with environmental stimuli was so strong that the boy blocked completely on several occasions. The general approach and manner of perception was characteristic of the schizophrenic, so that there seemed to be no suggestion of organic involvement. The original personality

integration and configuration had almost entirely disappeared from view, but one may suspect from the general tenor of the record that higher potentialities were still present.

Comment: This case was that of a 12-year-old boy who has been under constant observation for the past five years. On physical examination he showed infantile genital development. Repeated neurological examinations revealed no abnormal findings. The mental picture resembled that of the catatonic type of dementia præcox. The pneumoencephalogram revealed bilateral dilatation of the lateral ventricles, the left greater than the right. The electroencephalogram showed marked asynchronism of the waves of the two hemispheres and slow alpha waves. The psychological tests and the Rorschach examination indicated a schizophrenic reaction.

Discussion

During the past decade, a number of interesting contributions have been added to the subject of schizophrenia in children. In 1929, J. Kasanin and M. R. Kaufman[2] reported a study of 63 psychotic children observed at the Boston Psychopathic Hospital. At first, 21 of these cases were diagnosed dementia præcox, but after a period of study the diagnoses were revised so that only six remained classified as schizophrenic. The prognosis was considered "ominous" in these six cases. H. W. Potter[3] discussed schizophrenia in children in 1933 and reported six cases, the ages of which ranged from four to 12. Concluding he said, "Schizophrenia in children is apparently not as rare as it is thought to be." In 1935, L. Reznikoff[4] reported a case of schizophrenia in a child nine years and eight months of age. His patient complained of hearing imaginary voices and of seeing visions. Improvement occurred after six months, but the symptoms returned at the age of 13, and it was necessary to treat the child a second time.

Concerning the characteristics of the electroencephalogram in adult schizophrenia, Jasper, Fitzpatrick and Solomon[5] in a study of 82 cases said: "No single specific form of activity differentiates schizophrenia." Twenty-five per cent of their patients showed marked differences in amplitude, frequency, amount and form from homologous head regions. These investigators did find, however,

slow waves in 48 per cent of their cases. Jasper, Solomon and Bradley[6] found that the predominating characteristics of the electroencephalograms of five out of nine adult cases diagnosed schizophrenia were: "(a) continuous disorganized appearance with slow and fast waves mixed together; (b) dysrhythmia from bilateral homologous regions; (c) the most predominating feature of the abnormalities was slow waves from one or more regions of the head." P. A. Davis,[7] in an electroencephalographic study of 132 adult schizophrenic patients, found that the waves fell within three groups: "(a) essentially normal; (b) dysrhythmia (indistinguishable from electroencephalographs of individuals known to have convulsive disorders); and (c) choppy type which suggested possibility of brain pathology."

In 1940, Strauss, Rahm and Barrera[8] studied a group of children with psychiatric disorders. They examined 44 children with behavior problems of various types. Their ages varied from three and one-half to 13 years, and their I. Q.s averaged 95.1. Five types of electroencephalographic records were obtained: (1) normal; (2) borderline normal; (3) diffuse cortical dysrhythmia; (4) focal cortical dysrhythmia; and (5) doubtful. Sixty-eight per cent of the cases showed a definitely abnormal electroencephalogram; 16 per cent of the patients presented evidence of focal dysrhythmia. The largest percentage of cases was of the diffuse cortical dysrhythmic type. In only three of their cases was the electroencephalographic diagnosis confirmed by the usual neurological examinations. They concluded also that the coincidence of low intelligence and behavior problems should arouse a suspicion of organic pathology.

In the seven children here studied, the predominant features of the electroencephalographs were slow waves in all seven cases and asynchronism and slow waves in four cases.

At first consideration, this would seem to indicate some physiological disturbance of the cerebral cortices of all the patients. The evidence of similar changes in schizophrenic subjects, as offered by Jasper, et al., and Davis, would suggest that physiological abnormalities, as judged by changes in electrical potential of the brain, were a common feature of schizophrenia. Pneumoencephalograms apparently were not performed in the cases investigated by Jasper or Davis, nor are there records of detailed neurological

examinations. Therefore the following questions arise: (1) Is dementia præcox an organic disease of the brain specifically, or does it arise from organic disease of the brain? (2) Is it a psychogenic disease in adults but an organic one in children, or is it a psychogenic disorder in both children and adults? (3) Are organic changes of the brain mere coincidental findings in cases of dementia præcox? (4) Are the organic changes in the central nervous system predisposing or precipitating factors in the production of the mental disorder?

From the present study, none of these problems can be solved. In the first place, the series was small; certainly no broad inferences can be made from the study of seven cases. Furthermore, although in each of the patients there were abnormalities in the electroencephalogram suggesting organic disease, other workers have found similar changes in the brain potentials of schizophrenic patients. Hence no conclusions with reference to organicity can be made from the electroencephalograms alone.

The pneumoencephalograms afford more convincing evidence of organic changes in the brain. Dyke and Davidoff[9] have supplied standards for the normal encephalogram in their textbook. In the present series, five of seven cases showed definite though minimal changes in ventricular outline. The pneumoencephalograms were interpreted as indicative of cerebral hypoplasia.

Although the reliability of such type of testing as the psychological testing and the Rorschach interpretation is as yet not definitely determined, the writers believe it to be of value. In the seven cases, only one was definitely thought to be organic according to the Rorschach interpretation. In the performance tests the results were more variable but not conclusive.

Conclusions

1. Seven children varying in age from nine to 13 years, showing schizophrenic-like reactions, were observed continuously over a period of three to seven years.

2. Five of the seven cases showed evidence of organic diseases of the brain, probably on a hypoplastic basis as judged by the pneumoencephalograms.

3. All seven cases revealed the presence of slow waves in the electroencephalograms. Four of these had nonequivalence of both sides of the head.

4. Only one case was adjudged "organic" on Rorschach interpretation.

5. The authors cannot propose any definite conclusions regarding the problem of schizophrenia in children, but feel that further investigation along the same lines is indicated. This is in progress.

ACKNOWLEDGMENT

The authors acknowledge with gratitude the kind cooperation of Dr. George Kreezer, Dr. Bernard Pacella and Mr. W. E. Rahm for the electroencephalography; of Dr. Joseph S. A. Miller and Dr. Cornelius G. Dyke who corroborated the pneumoencephalographic interpretations; and of Dr. Zygmunt Piotrowski for his assistance with some of the Rorschach interpretations.

Children's Group
Rockland State Hospital
Orangeburg, N. Y.

REFERENCES

1. MacKinnon, I. H.: Noted from a discussion by MacKinnon on schizophrenia in children at the Psychiatric Institute in 1938.
2. Jasper, H. H., and Kaufman, M. R.: A study of functional psychosis in children. Am. Jour. Psychiat., 9, September, 1929.
3. Potter, H. W.: Schizophrenia in children. Am. Jour. Psychiat., 12, May, 1933.
4. Reznikoff, L.: Schizophrenia in a child. Jour. Nerv. Ment. Dis., 85, March, 1935.
5. Jasper, H. H., Fitzpatrick, C. P., and Solomon, P.: Analogies and opposites in schizophrenia and epilepsy. Am. Jour. Psychiat., 95, January, 1939.
6. Jasper, H. H., Solomon, P., and Bradley, C.: Electroencephalographic analysis of behavior problem children. Am. Jour. Psychiat., 95, November, 1938.
7. Davis, P. A.: Evaluation of the electroencephalograms of schizophrenic patients. Am. Jour. Psychiat., 96, January, 1940.
8. Strauss, H., Rahm, W. E., and Barrera, S. E.: Studies on a group of children with psychiatric disorders, I. Electroencephalographic studies. (New York State Psychiatric Institute and Hospital.) Psychosom. Med., 2:34-12, January, 1940.
9. Dyke, C. G., and Davidoff, E.: The normal encephalogram.
10. Lay, R. A. Q.: Schizophrenic-like psychosis in young children. Jour. of Ment. Sci., 84, January, 1938.
11. Despert, J. L.: Schizophrenia in infants and children. PSYCHIAT. QUART., 12, April, 1938.
12. Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: The likeness of the cortical dysrhythmias of schizophrenia and psychomotor epilepsy. Am. Jour. Psychiat., 95, September, 1938.

FACTORS IN PSYCHOTHERAPY: A PSYCHOANALYTIC EVALUATION*

BY M. RALPH KAUFMAN, M. D.

The problems of psychotherapy are exceedingly complicated. As defined by Warren,[1] psychotherapy is "the treatment of disorders by psychological methods; these methods differ widely, including waking suggestion, hypnotic suggestion, re-education, persuasion, psychoanalysis, Christian Science, etc." Hutchings' definition[2] is "treatment through influencing and regulating mental and emotional reactions." From these very definitions, one can glimpse the widespread and generalized field which is covered by the concept of psychotherapy.

For some years now, it has been the writer's pleasure to discuss with medical students the principles of psychotherapy. Many problems have arisen during the course of these discussions, most of them stimulated by the searching iconoclasm of the students. The preparation for these courses led of necessity to a rather wide reading on the subject, which embraced not only the so-called schools of Freud, Adler, Jung, etc., but also a study of the history and general development of psychotherapy. The proponent of each system makes definite claims that his particular approach produces therapeutic results. These claims vary from assertions of complete cure to statements of partial benefit; without doubt, the majority of them are sincere and are based on clinical observation.

This immediately brings one to the problem of what constitutes a therapeutic result in the treatment of the psychoneuroses and psychoses. Does the mere disappearance of the symptom constitute a cure; or are symptoms indicators of fundamental pathology which is not altered in any way by the so-called cure? One may use here for analogy the symptom of headache. There seem to be some 500 different known causes for headache. In the practice of medicine, one does not, as a rule, merely give aspirin to every patient who comes in complaining of a headache. One attempts through various diagnostic procedures to arrive at the etiological basis of the symptom. Brain tumor, neurosyphilis, frac-

*Read before the New England Society of Psychiatry, April 25, 1940, Hathorne, Mass.

ture of the skull, or astigmatism, all may present the symptom of headache. The treatment of each, if it is to be an adequate medical procedure, must be based on the etiological involvement. Treatment may under certain conditions be directed at the symptom rather than at the fundamental cause. Before this is done, the physician should, to the best of his ability, know what is the fundamental cause; he must also have arrived at a rationale as to why, under special circumstances, the treatment should be symptomatic rather than etiological. This is common and accepted practice in general medicine.

In psychotherapy, owing to many factors, not the least of which are great confusion and uncertainty with reference to the particular etiological factors, treatment for the most part tends to be symptomatic rather than basic. Most psychotherapeutic procedures are based on a theoretical substructure. If one believes that the psychoneuroses and psychoses are caused by focal infection, one then treats them by extracting teeth, tonsils, or puncturing and draining sinuses. If one believes that the thyroid is implicated, one may either feed the patient thyroid extract or perform a thyroidectomy. If, on the other hand, one's theoretical considerations lead him to the belief in psychic causation, the treatment may consist of one of various types of psychological approach, such as "talking the matter over," or advising against the impending visit of a mother-in-law. Those who believe in some form of specific physiological or somatic etiology are in a rather happy position, as far as their own peace of mind is concerned. There are many organs within the human body which may be operated upon without causing great physical damage to the individual. There are also many things which may be put into the body without greatly injuring the organism. It is only when these physiological theories call for drastic procedures that some begin to hesitate.

In modern psychology, a so-called dynamic-genetic etiological concept has arisen. This has stemmed in part directly from the psychoanalysis of Freud, and in part from the psychobiology of Meyer. These have had a tremendous influence on the psychiatric thinking of this country. Without attempting to enter into a detailed discussion of psychoanalytic concepts, the writer wishes to

present some of the basic principles of the theory of psychoanalytic psychology, and the procedures of psychoanalytic therapy.

Fundamental to all psychoanalytic thinking on personality is the concept of the unconscious. This unconscious is conceived of as a dynamic aspect of the human psyche, into which are repressed emotional experiences which for some reason cannot be tolerated in consciousness. These constellations remain in the unconscious, but are characterized by the fact that they retain the emotions originally experienced. Another characteristic is that these emotional experiences constantly seek expression and require a constant pressure on the part of the repressing forces to keep them from reappearing. The unconscious is also the repository of the instinctual drives and needs of the individual. The motivating forces of personality function are for the most part unconscious and not subject to conscious control. Psychoanalysis is essentially a psychology dealing on the one hand with the instinctual drives of the individual, and on the other with the various forces in the environment, culture, and within the individual, which tend to allow or disallow the gratification of these drives.

Psychoanalysis places particular emphasis upon the first years of life, maintaining that the patterns of behavior laid down up to the sixth year are of utmost importance to the personality structure and to the subsequent neurotic or psychotic manifestations. Certain biological needs are common to all individuals, and these center in infancy upon feeding and secretory-excretory functions. Some needs are satisfied within the organism independently of the environment. Others, such as feeding, are dependent for their fulfillment upon the environment, as the mother or the nurse. These soon become intimately related to and conditioned by the environment; and in that way, they become secondarily charged with emotion. The infant reacts to the gratification of these needs with emotional responses. Specific emotional significance is attached to an individual in the environment in accordance with the rôle he plays in the fulfillment or frustration of these needs. The gradual adaptation to reality begins early. Should the gratification of any of these needs be frustrated, as in weaning and habit-training, the individual reacts with aggression to the frustrating experience. This aggression may be expressed; or, owing to cer-

tain factors, for instance the fear of punishment, it may have to be repressed. The discipline to which the infant is subjected is first reacted to in terms of the external objects exercizing the discipline. Soon, however, through the complicated process known as identification, the infant or child takes over the "forbiddance," and there is less need for an external authority, since the instinctual need concerned is then restricted by the child itself. One aspect of the personality, the ego, gradually becomes the medium through which reality on the one hand and instinctual needs on the other are evaluated.

The concept of conflict has had an important influence in psychiatry, and it may be seen from the above schematization how conflicts may arise, either between the environment and the individual or between various elements within the individual, often between the instinctual needs and the inner forbidding forces which constitute the "conscience." These conflicts may be solved in "normal" ways or by means of a neurosis. According to psychoanalysis, neurotic symptomatology represents essentially an attempt within the individual to solve a conflict along the lines of a regressive compromise, so that a neurotic symptom contains within itself at least two elements: a partial gratification of the instinctual need, and a partial acceptance of the forbidding forces which prevent gratification of the need. One of the major contributions of psychoanalysis has been this realization that neurotic and psychotic symptoms are not fortuitous or haphazard, but are of particular significance to the individual who is suffering from them. Since they are a compromise resultant from conflicting forces within the personality, they constitute an attempt to achieve an equilibrium. In other words, they are the best possible solution under the particular circumstances.

Among the various emotional experiences of the individual, anxiety plays a special rôle. A number of theories have been posited relating to the origin of anxiety and its function in the life of the individual. It would take one too far afield to discuss in detail the changing hypotheses in psychoanalysis concerning this aspect of the problem. Today, anxiety is considered as playing a fundamental rôle in the etiology of the neuroses. Under what circumstances does anxiety arise in the child? Usually in any situation

where the child feels threatened. These threats may come from the external environment in the form of a parent, nurse, teacher, or sibling, and usually are associated with some form of denial of an instinctual need or desire. The external authority has the right to punish the child for the breaking of any taboo. Therefore, when the need arises under certain circumstances, its gratification is inhibited by a fear of punishment. However, this threat does not always remain in the external world. Through a complicated mechanism, associated with identification, the threatening figure may become internalized, and when the particular need or desire arises, the threat of punishment is usually associated with the feeling of guilt which is the operation of the function of the superego. In any given danger situation, therefore, anxiety may be precipitated. This anxiety fulfills a double function: At the same time, it acts as a sort of signal of impending danger and serves to call into play forces which inhibit a desired gratification. This formulation, of course, represents an oversimplification of the actual state of events.

One must, however, differentiate between anxiety and fear. Fear is a subjective experience usually related to an impending danger, consciously perceived in the outside world. Anxiety, on the other hand, may and usually does arise in relation to a dangerous situation the source of which is unknown. An unconscious striving, the fulfillment of which threatens the individual in some way, may be kept from consciousness by an outbreak of anxiety which brings the repressing forces into play. In this conflict situation, there is a struggle between repressed needs and other forces within the personality. So long as this conflict is not solved, the individual may be subjected to anxiety. A partial compromise solution of this conflict in the form of a neurotic or psychotic symptom may temporarily establish a state of equilibrium at a regressive level; and the anxiety may then subside, to reappear only when the equilibrium is upset.

A simple illustration of the mechanism involved may be observed in the phobias, where as long as the patient avoids the threatening situation—e. g. the street or an animal—he does not suffer from anxiety. Should, however, the instinctual need grow stronger, or the repressing forces weaker, the patient will show

an increase in symptomatology, so that the originally localized phobia situation becomes more and more generalized. The symptoms which are at first related only to a fear of crossing an open place may eventually incapacitate the individual to a point where he has to remain locked up in his own room. The obsession-compulsion neuroses offer a good example of a similar type of mechanism. As long as the ritual is carried out, anxiety is minimized. In the long run, however, there is an increased need for ritualized behavior, since the unconscious striving threatens to erupt into consciousness; and more and more symptoms must be constructed to evade knowledge of the forbidden wish. In the psychoses, certain panic states are characteristic. The overwhelming anxiety suffered by the individual may be an indication of the force of the threatening unconscious needs. So-called homosexual panic is a good example. Freud has pointed out that many of the symptoms seen in the paranoid psychoses and the schizophrenias are in reality attempts on the part of the individual to make a new adjustment; one may even go as far as to say they are attempts at self-cure.

At times the content of delusions clearly shows both the unconscious instinctual needs and the forces in the environment and personality which are directed against their gratification. Conscious and unconscious feelings of guilt are often associated with anxiety. The mechanism of the guilt reaction again is exceedingly complicated. However, feelings of guilt may arise whenever the individual does, or wishes to do, something which is forbidden. This wish need not be conscious. Aggression and hostility against an object bring with them the fear of retaliation. The need to express hostility may be repressed, but may nevertheless be associated with a marked sense of guilt and a need for punishment. This concept is of particular importance when attempting to understand some of the more primitive psychotic manifestations, particularly the depressive phase of manic-depressive psychosis.

Owing to a number of factors, certain periods of life—childhood, the so-called latency period, puberty—are of tremendous importance to the individual from both the somatic and the psychic points of view. New demands, both instinctual and environmental, arise at these periods and the individual attempts to meet them. Should

these demands be of such nature that the resultant conflict cannot be solved within the framework of normal personality reaction, the result may be a neurotic or psychotic episode, which is essentially an attempt at solution of an insoluble problem.

Psychoanalysis has stressed particularly the emotional relationships which evolve during the earliest period of the individual's life, and has attempted to show that these emotional relationships, once laid down, tend to form patterns which repeat themselves whenever one or more factors of a particular pattern appear. Thus, early hostility to the father may determine the individual's subsequent emotional reactions, at both a conscious and an unconscious level, to figures who have the attributes, either real or in fantasy, of the father. In the same way, the pattern laid down by the child for the handling of his instinctual needs may determine not only his personality structure, but the type of neurotic behavior which he may subsequently show. From this point of view, the determining components exist within the personality from a very early time of life. However, during the subsequent course of the individual's life, various factors, either increased instinctual needs or environmental stress, may act as precipitants of a neurotic or psychotic picture. This point of view does not in any way minimize the important rôle played by the current life of an individual in the neuropsychoses. It is desirable to emphasize this, since some nonanalytic writers have insisted that the psychoanalyst entirely disregards such current problems.

It is of course difficult within a limited space to do more than touch the highlights of a complicated psychology such as psychoanalysis. On the basis of empirical clinical experience, Breuer and his coworker Freud built up a therapeutic system based on the use of hypnosis and the revivification of buried or repressed traumatic experiences. At this time, Freud gave full credit to Janet, who used what we recognize today as essentially a method for emotional catharsis, which was effective for symptomatic relief in the hysterical neuroses. Enlarging experience soon showed Freud several things: That these traumatic events which he at first implicated as the main etiological factors were not confined to any single experience. His patients, while under hypnosis, began to uncover experiences which went back into their earlier lives. These seemed

to be of the same pattern as the later traumatic incidents. He discovered also that many of these so-called memories were not memories of actual happening, but were related primarily to the fantasy life of the individual. Further, many of his patients he found to be only temporarily symptom-free; and, under new stresses and strains, a repetition of their symptomatology would occur. This led him to the development of a "free association" technique, which was an attempt to arrive at the unconscious thoughts, fantasies and conflicts of the patient without the use of hypnosis.

In addition, he made a fundamental discovery for psychotherapy. He became aware of the fact that the relationship between patient and physician was of tremendous importance in the psychotherapeutic procedure. In itself, this discovery was not a new one. The perspective, however, in which he placed it and the use he made of it were completely new. This relationship, which is called transference, is basically a repetition of the individual's emotional attitudes toward the most significant figures in his early environment, particularly the parents. These attitudes, developed during childhood, may for the most part be unconscious and are essentially of an ambivalent nature. To the child, the father represents an omnipotent and omniscient individual, who is responsible for his well-being and is a source of love and affection. In addition, the father is an authority who must be obeyed. As a source of discipline, his rôle is that of one who may also frustrate and punish the expression of instinctual needs. It is because of this that certain aggressive, hostile feelings are directed against him. The same is true of the mother. During the treatment, these early emotional attitudes are projected onto the therapist and arise independently of the current situation. Reality may color this relationship to some extent, but the transference emotions are repetitions of childhood patterns. The transference relationship occurs regularly in all therapeutic settings, and is not peculiar to psychoanalysis.

In the positive phase, it has certain characteristics. The patient overevaluates the therapist, looks upon him as an all-wise authority, and may even accept every suggestion without the slightest difficulty. A relation of affection exists in which the patient

feels secure. It is during this period that many of the presenting symptoms disappear.

Due partly to the ambivalence of feelings and partly to the frustrations and lack of gratifications inherent in the therapeutic situation, the positive feeling may soon change into a hostile one, which Freud has called the negative transference. During the phase of the negative transference, the attitude of the patient toward the therapist changes completely; instead of overevaluating, he may begin to undervalue him. The latter's advice becomes invalid. His therapeutic efforts are rejected, and hostility and aggression may be frankly expressed. Symptoms which have previously disappeared may now return. The psychoanalyst is cognizant of both aspects of this transference relationship and consciously uses it as a fundamental instrument of his technique.

A knowledge of the existence of transference is necessary if one is to evaluate adequately any psychotherapeutic technique. The psychoanalyst's attitude is one of neutral sympathy. He does not attempt to judge or to evaluate the individual's reaction from a moral or personal point of view. It is this atmosphere of calm reassurance which plays a great part in the ability of the patient to bring gradually to the surface many of his problems.

An accompanying phenomenon which psychoanalysis has described is that of countertransference. This is the emotional attitude which the therapist develops toward the patient. The physician is a human being, with biases, prejudices and problems of his own, which he brings into the therapeutic situation. These attitudes may be reflected during the course of the treatment in relation to the patient. One may, for instance, attempt to inculate into the patient one's own philosophy of life, or one's attitude toward moral problems. Certain emotional conflicts within the therapist may scotomatize him to a similar conflict in the patient. On the other hand, the therapist may see in every patient a reflection of his own unconscious problems. He may have an inner need for constant praise from his patient and may not be able to tolerate failures. He may suffer inability to withstand the hostility directed against him by the patient. The physician, as a rule, tends to accept only the positive attitudes of his patient, rejecting the negative ones. Unless he is aware of the transference process, he

may personalize the emotional attitudes of his patients toward him, accepting them at their face value, without realizing that actually, for the most part, the emotional attitude is a projection of the patient's fantasies, and that unless the physician gives the patient a realistic basis for such attitudes they have actually little to do with him as an individual.

This phenomenon of countertransference has major implications for psychotherapy. It may determine the therapist's attitude toward the problems of the neuroses or his philosophical bias as to what constitutes adequate treatment. Certainly it will be of determining importance with reference to the type of patient he may be able to treat adequately. A common example of the attitude of the average physician toward such problems is clearly exhibited in the outpatient departments of most general hospitals. One of the reasons for didactic analysis as a preliminary requirement for the practice of psychoanalysis is based upon knowledge of this problem. Such analysis enables a therapist to come face to face with his own problems and to a certain extent to solve or objectify them in order that they may not interfere with his treatment of his patients. Meyer has recognized this, and his emphasis on the need of a personality study of the psychiatrist is the outcome of this point of view. It is presumed that through this personality study the psychiatrist will obtain a knowledge of his own emotional problems and a new perspective toward his personality, in the light of which his biases and prejudices may be made not to interfere with his adequate functioning as a therapist.

Free association in psychoanalysis and in other forms of therapy is a tool designed for many uses. With the patient relaxed and noncritical, the free association may lead to fantasies, ideas and emotional experiences of which the individual has not been aware before; it is designed in psychoanalysis particularly with this purpose in mind. However, it is not in itself the main therapeutic measure. Verbalization serves many functions, and in analysis this verbalization is in part an attempt to keep the individual from acting out in the motor field certain of his emotional needs. It can, however, also serve the purpose of rationalizing these needs. A slogan or formula may hide from the individual the actual emo-

tional situation which the formula symbolizes. A good many of the so-called insight therapies remain at this level.

The psychoanalyst not only uses the material brought out by free association, but he is also keenly aware of the points at which the free association ceases. It is when a thought or idea is suppressed rather than spoken that one often obtains a hint of something which may be painful or unpleasant for the patient to talk about. In the original concept of psychoanalytic therapy, emphasis was laid only upon the bringing to consciousness of unconscious material and its interpretation. This is still important today. Emphasis, however, is now laid also upon the phenomenon of resistance, and those factors in the personality which tend to keep instinctual needs from consciousness are analyzed. The reasons why the patient resists the coming to consciousness of certain aspects of his emotional life are worked through. Usually when this is accomplished, the material appears either in the free associations or in the dreams of the patient.

There may be associated with the phenomenon of resistance a disappearance of symptoms. It is as if, rather than face his unconscious, the individual temporarily gives up his neurosis. Gradually, as the process of analysis goes on, more and more areas of the patient's personality are explored, gaps in his memory are filled, unconscious material is brought to the surface. The interpretation of relationships forms a most important part in the psychoanalytic technique. These relationships and patterns of behavior are interpreted through the medium of the transference reactions. The current patterns of behavior, the actual life situation of the patient, are important in every analysis. No analytic procedure is complete until these are understood in the light of the patient's total personality. The final phase of every psychoanalytic procedure is the resolution of the transference relationship in terms of the reality situation.

Of what value is this review of psychoanalytic psychology and therapy for the understanding of other psychotherapeutic procedures? Before attempting an answer to this question, it will be pertinent to note the following excerpts:

"The first step on the part of the medical man, is to gain the

confidence of the patients by kind treatment, and a solicitude for their welfare. These are soon perceived and properly appreciated. To engage their attention on some new object either by affording them useful employment or attractive recreation, is the next step to be pursued. . . . But with others of this rank, it is a task of no ordinary difficulty to rouse the patients to any species of exertion, mental or bodily. This particularly is the case where the disease has been of long standing; the mind having become habituated to one train of thinking, and the body to indolence, the greatest repugnance to any exertion is felt. In some constitutions nothing but the most determined perseverance can overcome it. The great means of accomplishing this, or indeed, of influencing the conduct of the patients in any other respect, is by ascertaining what they particularly like and dislike, and then granting or withholding the indulgence, according to their behavior. Very few persons arrive at the period of life at which insanity comes on without having acquired certain tastes and habits. It is of the greatest importance that these should be ascertained in each individual patient. They are the lever, and frequently the only lever, by which the moral man can be moved. When the bodily health is restored, any little things which the patient really enjoys should be withheld, and only granted upon his complying with certain conditions and withdrawn on their being broken. The medical attendant ought to be ingenious in finding out the pecularities, and to be firm and kind in the treatment which he founds upon them. He ought fully to explain to the patient the reasons for his conduct to him; and endeavour to impress upon the mind, that any other mode of treatment would be a breach of duty on his part, and that the deprivation is painful to him, but essential to the patient. In many cases, where the total indifference of a patient prevents this mode of treatment being used, the breaking in upon his habits has a similar effect. . . .

"Considerable tact is required in adapting the particular kind of occupation to the tastes of the patients. They are usually more easily induced to work at the trades to which they have been brought up, than to turn their attention to pursuits entirely new. Most men seem to have a natural fondness for farming and gar-

dening, and these occupations have this great advantage, that there are certain portions of the labour in them, in which a violent or suicidal patient may be employed, without being entrusted with any tools by which he might either injure himself or others. But so important do I consider the diverting the mind by employment, that where the patient cannot be induced thus to occupy himself, or where the occupation is too mechanical to keep the mind interested, I do not hesitate, with proper precautions, to intrust him with tools, even where an inclination to suicide or to violence exists. . . .

"Of course, many will be found to whom such an employment would be irksome; but, whatever be the rank of life, or the difference in outward circumstance, the man is still the same being. He feels pain when deprived of the comforts which he has been in the habit of enjoying—he is to be won by kindness, and he is offended at harshness or want of courtesy. The being excluded from the society of all whose good opinion is valued, begets in the insane, as it would tend to do in the sane, a habit of giving utterance to momentary feelings, without considering their propriety. And with both, where the mind has no opportunity of employment on objects of importance, it will either busy itself about trifles, or sink into apathy, or allow itself to wander unchecked in idle reveries. . . .

"In a well-regulated institution, every means ought to be invented for calling into exercise as many of the mental faculties as remain capable of employment. We must remember, that the happiness of man, whatever be his situation in life, consists in the proper and harmonious exercise of all his powers, moral, mental, and physical. . . . For persons in the higher ranks of society, a mansion should be provided, with park, woods, lawns, hot-houses, gardens, and greenhouses. It should be fitted, internally, with every convenience and luxury for the gratification of the taste. Science and the fine arts ought to be pressed into the service of stimulating the dormant faculties to healthy exercise. There should be a music-room, which the patients of both sexes should daily have the privilege of using; and one evening in every week should be specially devoted to a dress-concert or oratorio, to which all, in a fit state to attend, should be invited. Such an association of patients, of the two sexes, would have a very happy influence

on both. . . . There should be a modeling room, and a studio, where those who have a taste for the fine arts should have an opportunity of receiving weekly instruction. . . . The library should be well furnished; but, of course, care and discrimination would be required in the selection of books, adapted to the particular habits, and to the states of mind of the patients. . . .

"There would then be an evidently useful object in their employment. . . . Few minds are so constituted as to be able to employ themselves merely from an abstract notion, that activity is conducive to happiness. . . . They are frequently asked to work, without knowing for what purpose; and as might be expected, such occupation becomes tedious, and is at length refused."[3]

The above lengthy quotation, aside from its somewhat quaint phraseology, may be taken as an adequate statement of the present-day therapeutic approach in most hospitals. Indeed some of it has such a familiar ring that one might suspect the quotation of coming from a recent psychiatric journal. Actually it was published in London in 1838, in a book by Sir W. C. Ellis.

Since in the past hundred years our psychiatric knowledge has advanced tremendously, particularly that aspect of it which has to do with our understanding of the psychological factors which play a rôle in the neuropsychoses, it would seem justifiable to maintain that the rationale of our therapeutic procedures should keep pace with this increase in knowledge, and that we should not follow a rutted path simply because of its practical value. We should attempt, if possible, to understand the dynamics of our psychotherapeutic procedures in the light of our new knowledge. The writer would emphasize that he is in no way seeking to minimize the value of other, nonanalytic forms of psychotherapy. Even further, it will be understood for the purposes of this discussion that there may be many other, nonpsychogenic factors involved in the etiology of the neuroses and the psychoses. Constitutional factors have always been particularly stressed by Freud himself. The rôle of heredity should not be minimized. However, in spite of these things, we still work psychologically with most of our patients. Even individuals with pneumonia may be soothed in a delirium by an understanding nurse without the use of a sedative. This does not imply that sedatives are not effective.

The hospital and the hospital organization play an important rôle in psychotherapy. It is not so rare an experience that excited, anxious, or depressed patients may show an immediate relief from symptoms on entering a hospital. There are numerous factors involved, not the least of which may be relief from responsibility and the demands of life. In other words, one of the components which has led to conflict within the individual has been temporarily minimized. Such "cures" are usually temporary, since the underlying conflicts are of a dynamic nature; and the solution by hospitalization is as a rule not sufficient. One sees patients, however, who under hospital routine and conditions may make what we call a satisfactory adjustment. Such patients soon after leaving the hospital will experience what appears to be a recurrence of illness.

There are many procedures within the hospital routine which are primarily of psychotherapeutic value. The part played by occupational therapy has been emphasized from many points of view. The task upon which the patient engages may have, among other things, a definite symbolic significance, and allow for the gratification of certain repressed impulses, for instance partial components of infantile sexuality. Thus a patient engaged in clay-modeling may be sublimating with the permission of the therapist some of his earlier tendencies to play with mud or feces. The products or designs of sculpture often symbolize a patient's fantasy. For example, a schizophrenic patient, busily engaged in modeling bizarre clay ash trays, produced a series of objects which on even superficial examination were seen to represent the vulva and pubic hair. It is of interest that this particular patient's delusions consisted in part of ideas concerning tremendously exaggerated male and female genitals. Other patients may represent in paintings and drawings elements of their conflict and work out solutions at a sublimated level.

Another aspect of occupational therapy is that it allows a patient to mingle again with the group, to be accepted, and to carry on relationships with other people, patients and workers, under conditions of minimum demand. Frequently, the relation with an understanding occupational worker is more important than the actual type of work. To a great extent, the treatment by various

nurses and attendants is significant. The hospital staff, from superintendent to attendant, forms in a way a new family. To the patient's unconscious, nurses and attendants may frequently represent parents and siblings, who are cooperative, sympathetic, and anxious to help, and there may ensue a minimization of the rivalry which the patient has had to face in real life. Another phase of the psychotherapy which a hospital offers is the relationship between patients. If one works intensively with hospital patients, one soon discovers that a good deal goes on between the patients of which the physician is not aware. Patients make confidants of each other. They may "gang up" on one or the other. They gossip. They tell each other their troubles. They may even make suicide pacts or plan methods of escape, which remain at the level of verbalization. Here again, one sees the acting out and temporary solution in a protected environment of conflict situations which the patient cannot handle outside. Athletic activities which allow for competition and for the expression of various hostilities and aggressions may in themselves also have therapeutic significance. Various social activities, dances and movies, all present to the patient a miniature world free from the demands made by the world outside. The secondary gain of the illness may be worked out in relation to these aspects of the hospital routine; in this way, some patients accept the hospital as a therapy as long as they can remain within hospital walls.

At this point, it should be stressed that each patient must be treated and understood as an individual, and that hospital activities should not be routinized and regimented. Not every patient is helped by occupational therapy. Many patients may resent the level which this work represents. The same is true of social activities. The shy, awkward schizophrenic, whose problem may lie within the sphere of interpersonal relationships, particularly with members of the opposite sex, may have an increase in symptoms if forced routinely to attend such functions. The common practice of having only male nurses with male patients may have an important repercussion on some patients. Frequently one sees this in the accusations made by patients against nurses, and latent homosexual conflict is stimulated rather than diminished by such contacts.

One may demonstrate that in certain aspects of the various departments of the hospital, such as hydrotherapy, the treatment is primarily of psychological, and only secondarily of physiological, importance. Particularly in hydrotherapy, one should bear in mind the part played by perceptions of tactile sensations in the pleasure-seeking activities of every individual. The skin and musculature are a source of erotic gratification. The prolonged warm bath may fulfill for the patient certain primitive sexual needs. Empirical clinical observation shows in some patients the fantasy of a return to the rest and quiet of the mother's womb, gratified symbolically under these conditions. Massage and various other stimulating forms of psychotherapy may also serve certain psychosomatic needs in the individual.

In recent years, various hospitals have pointed with pride to their "beauty shops." These certainly are of direct psychotherapeutic value, especially in relation to the narcissism underlying pride in physical appearance, which is such a marked component of the feminine attitude.

In all forms of directed psychotherapy, the work of the individual physician with the patient is of primary importance, irrespective of the technique employed. There are many procedures favored by physicians.

These techniques may be divided into two broad groups. One has as its main purpose the allaying of emotional turmoil and anxiety. Various procedures may be employed with this aim in mind. Hypnosis, during which suggestions are given that the symptoms disappear and that the patient feels better, is a method which in the past enjoyed considerable vogue. Various forms of reassurance, from the denial of the fact of illness by the physician to the statement that "in a little while everything will be all right," suggestion directly or indirectly given, and forms of authoritative pronouncements, fall into this category. Attempts to interest the patient in different types of work, social activity, etc., to distract him from his symptoms, also fall into this category.

The main purpose of this type of technique seems to be to help the patient in one way or another to repress or to push aside the symptoms. The mechanisms involved fall into several categories. The positive transference relationship usually plays a rôle, the

patient accepting the reassurance partly because of the authority of the physician, partly because there is a decrease of guilt and anxiety. That part of the personality of the patient which is struggling against the illness is reinforced in this way. There is no attempt made to understand what underlies the symptom, but rather all therapeutic efforts are designed for denial and minimization of illness. Various forms of environmental shifting may also fall into this classification. Minimizing the demands of the environment, helping the patient by obtaining employment for him, decreasing pressure by economic help, may diminish the force of the precipitating factors in the conflict, and may give the patient an opportunity for reorientation.

The second category of therapeutic techniques might be called the unmasking or uncovering type. This essentially amounts to bringing the patient to understand factors and conflicts of which he may not be aware and to helping him toward a new type of solution, either by direct advice or by increasing his ability to tolerate conflict at a conscious level. The majority of modern psychotherapeutic techniques, including psychoanalysis, are based upon this approach.

There are a number of reasons why the classical technique of psychoanalysis has not been made available for the treatment of the psychoses. Freud and his earlier coworkers emphasized the fact that the procedure was particularly suited for application to the psychoneuroses. Both theoretical and practical considerations are involved. For the most part, the clinical material available to them in private practice was essentially confined to the neuroses, also the technique lent itself particularly to work with patients who cooperated and who recognized their need for therapy. On the technical side, Freud differentiated between the neuroses and the psychoses in terms of transference neuroses and narcissistic neuroses, including in the latter group those we today label schizophrenias and manic-depressive psychoses. In a limited sense, this division still holds good. Because of these factors, for many years the psychoanalysts were wary of attempting to treat any of the major psychoses. Psychoanalysis was used in this connection only as a research instrument to investigate the meaning and content of the psychotic reaction, rather than as a therapeutic pro-

cedure. Certainly for the vast majority of the so-called functional psychoses, psychoanalysis even today is not the method of choice. There have been some attempts to employ analysis, with or without modification, in the treatment of selected cases of schizophrenia and in the manic-depressive group. The results reported vary from success in individual cases to complete failure. The field has as yet not been thoroughly explored.

It may be stated that this method is applicable to certain carefully selected patients, if employed by analysts of wide experience in the field of clinical psychiatry. The principal difficulty lies in the inaccessibility of the patient. That this inaccessibility is subject to modification is well known in psychiatry, since it is an everyday experience in the handling of patients in hospitals that, to varying degrees, many do establish contact with the physician and the hospital organization. For many years it has been stated with justification that psychotics, particularly schizophrenics, have broken with reality. This is undoubtedly true. However, just what this break with reality consists of would not seem as yet sufficiently clear. The psychoanalytic formulation, epitomized in the statement that in psychosis the conflict is between the ego and reality, has significance but certainly is not the final answer to the problem. Reality relationships are distorted. Whether this distortion represents a complete break with reality is a question. It is the writer's impression that in many instances the possibility for contact with reality is there, and that what a psychotic does is to add to or subtract from certain aspects of reality, while retaining the ability to evaluate those aspects of reality which are not directly involved in his conflict situation. The physician in his relations to the patient is usually involved in both aspects of the attitudes to reality. In the experience of many, the psychotic, even the schizophrenic, at his most deeply regressed level still retains a capacity for establishing transference relationships. These relationships differ in degree and perhaps in lability from those established by the psychoneurotic. However, the important thing is that this capacity is retained and that relationships are possible. Transference manifestations may be related to earlier aspects of personality organization and to more primitive needs,

and therefore may be more tenuous and dangerous, hence may require a different "handling." They are, however, basic for any working rapport with the patient.

Since the psychoanalytic technique is not the method of choice, it is important to use other techniques in the treatment of the psychotic patient. In American psychiatry, the procedures based on Adolf Meyer's psychobiology are utilized primarily in this treatment. Here an attempt is made to bring together as many facts about the patient's life as possible; to trace through his development and habit formation; to evaluate the methods which he employs for the handling of emotional crises; to give the patient a new perspective on his personality reactions. A certain element of advice and reorientation is always present in this technique. In addition, manipulation of environmental factors, through either hospitalization or change in the outside world, is attempted. By and large, this technique does not extensively stress the unconscious factors, but works with conscious elements and perhaps with certain personality aspects of the individual which are more or less near awareness. Emphasis is laid on the genetic development of the individual, as understood at that level. In practice, it is an effective therapeutic measure, particularly in the handling of psychotic problems.

There are several dynamic factors in this therapeutic procedure. The relationship of the patient to the therapist is of paramount importance; however, it is not utilized by the physician in the same way as is transference in the psychoanalytic setting. Nevertheless transference, particularly on the positive side, is an important factor in the therapy. The patient accepts or rejects advice, not only because the advice is valid or invalid, but primarily because of his relationship to the adviser. The type of insight gained in this sort of evaluation of one's personality is valuable. It tends to give the individual a new perspective, but also a new series of rationalizations. Here again the most important issue is not so much whether what the patient sees is basic, or even altogether true; it is that a new orientation and a system which offers a possibility of solution of conflict are given to the patient.

One must bear this point in mind particularly in evaluating certain institutions within our culture, to be referred to later, which have a psychotherapeutic value.

Another element which enters into this therapeutic approach is that of emotional catharsis. Directing the patient, or giving him an opportunity, to discuss certain of his emotional problems, allows for a discharge of the repressed emotion. This in itself is of value in alleviating the need for symptoms.

A number of other schools of therapy, notably those of Adler and Jung, employ this uncovering technique. These systems each deal with the personality at a different level. The Alderian system, emphasizing particularly as it does a rather simplified formula for the genesis of neurotic and psychotic behavior, namely the reaction to feelings of inferiority with the resultant strivings for superiority and a goal in life, also the rôle of patterns laid down in early childhood (so-called life-style), offers the patient a combination of catharsis, rationalization, and insight to a certain level. Here again the function of transference is important, even though the therapist does not emphasize it.

The Jungian system is somewhat more complicated, and is really a combination of "unmasking" and "masking." The rôle of conflict in the individual and the ontogenetic factors of personality development are stressed. At the point, however, where the Freudian begins to emphasize early interpersonal relationships and instinctual demands, the Jungian digresses and shifts emphasis to a somewhat mystical "collective unconscious." It is at this level that an effective rationalization and flight from guilt with a consequent allaying of anxiety takes place, since it is easier, for instance, for a patient to accept the forbidden sexual relationship toward a real mother if it is posed in terms of a cosmic conflict, depersonalized and inherited via the route of the collective unconscious, which is phrased in terms of archetypes.

Another form of so-called insight therapy, hypnosis, seems to be regaining favor. In this use of hypnosis, an attempt is made to direct the patient toward reviving memories which have been repressed. These incidents with their attendant affect are brought into consciousness and relived. In certain types of neuroses, particularly in conversion hysteria, there is a definite beneficial effect

on the symptoms. However, these results would seem no happier in the long run than those of Freud when he first used this technique. Transference plays a tremendous rôle here, too. The individual does not really accept and digest the traumatic experience, but is enabled to tolerate it temporarily with the aid of the therapist and suggestion by the therapist. This technique has really little in common with psychoanalysis.

There is a special need at this stage of our psychiatric endeavors, when an impetus has occurred in the direction of chemotherapy in the form of shock treatment, to attempt to maintain balance. One of the difficulties encountered when any new therapeutic agent seems to show great promise is the tendency to concentrate on the use of that agent with a sidetracking of all other forms of therapy. During the past several years there have accumulated in the literature many reports concerning the therapeutic value of the various forms of shock treatment. It is of interest to note that, at the high tide of enthusiasm, the emphasis was primarily upon the value of the drugs employed in terms of physiological changes. Opportunity for further clinical observations resulted in a trend toward the increased evaluation of the possible psychological factors involved. The relationship between the psychiatrist and the patient at varying stages of the shock treatment began to be discussed. Some workers now believe that the fundamental therapeutic value lies in the increased rapport between the patient and the therapist immediately following the shock, with the use of the opportunity to understand with the patient the various factors entering into the psychoses. Others caution against any discussion of the delusions and trends, hoping to help the patient repress the "pathological" symptoms.

The question has been raised of the rôle played by anxiety in this treatment. In general, one may state that, irrespective of any physiological factors involved, it is important to realize that certain psychological factors are dynamically effective in all forms of shock treatment. In the light of our knowledge of the great part played by anxiety in the genesis and continuation of the psychoses and the neuroses, it seems probable that any treatment involving the precipitation of immense quantities of anxiety must have a psychological effect upon the patient.

One may presume that in certain individuals this anxiety may act as the lever of a mechanism leading to the temporary repression of symptoms. Some patients seem to react to the convulsion as if they were being punished, and perhaps this punishment serves an unconscious need of the individual, allowing an expiation of his sense of guilt. The writer has observed this mechanism in several patients, who asked for a repetition of the treatment. On examination, contrary to superficial expectation, the reason for requesting continuation was not that it made them feel better, but that it symbolized punishment to them. In one individual, it was definitely used as a punishment which alleviated a feeling of guilt related to a current masturbation conflict. Other patients show an intensification of their homosexual patterns, particularly in the period immediately following the convulsion. They seem under these circumstances to be able to express their homosexual needs more freely and openly, and may even make homosexual demands on the physicians and nurses.

A characteristic response which has been reported particularly in relation to metrazol, and which the writer in collaboration with others had observed in carbon dioxide-oxygen therapy some years ago, is the persistent fear of death. Some reports on this aspect of the problem minimize this fear as of little therapeutic import. However, it was the writer's distinct impression that in certain instances it played a definite rôle in the disappearance of symptoms. On the psychological side, the whole of the so-called shock therapy is related to various other forms of "shock" attempted in psychiatric practice over many years. The rationale behind these various attempts has always been phrased in terms of "bringing the patient back to reality."

The various elements in this form of treatment, which are essentially of a massive psychotherapeutic significance—anxiety, fear, fear of death, punishment—are factors which may force the patient into giving up his symptoms temporarily. In addition, the relationship between the physician and the hitherto inaccessible patient may be deepened and possibilities for transference relationships established. In many instances, it is perhaps this relationship which is the main therapeutic agent. Irrespective of the nature of the ultimate pharmacodynamic mechanisms involved, all

forms of shock treatment have a definite psychological aspect, and in this field particularly, intensive investigation along psychological lines will yield valuable results in the evaluation of the dynamics of psychotherapy.

In an earlier part of this paper, allusion was made to the various institutions in our culture which serve, among other purposes, as psychotherapeutic agents. Man, during the course of his evolution, has created and utilized certain cultural patterns, such as religious beliefs, which, regardless of their ultimate truth or falsity, also serve the purpose of minimizing and allaying his anxiety.

Studies of the genesis of some of the more esoteric religious cults readily reveal the psychotherapeutic elements. Conflicts with the father, fear of death, the need for a stronger discipline in relation to forbidden instinctual demands, may be projected into religious beliefs. One of these systems, for instance, involves a striking denial of reality, which if perceived in a single individual might constitute a schizophrenic reaction. When these beliefs are reinforced by an identification with a group, they allow for a working through of various anxiety situations within a permissive social group. All psychiatrists are familiar with the rôle which may be played by ascetic ideals within a religious community in the repression of instinctual demands. Emotional catharsis, expiation, and the sense of being forgiven by an omnipotent father allow for the attenuation of guilt feelings. The problem of the therapeutic rôle of these types of belief in a culture has as yet barely been touched upon. Nevertheless, many psychiatrists consciously use these agencies in their attempts at therapy.

Cultural patterns not only serve as disciplinary mechanisms, but also under certain circumstances may be permissive. Aggression, unless attenuated and sublimated, must generally be under control. Under a given constellation, however, aggression becomes not only permitted, but idealized. In war, killing, of the enemy of course, makes a man a hero, acclaimed by the multitude. In peace, it makes him a murderer. In certain nations, however, the various instinctual needs of the individual seem to have been taken into consideration almost consciously: Sadism, hatred, and aggression are given full play for purposes which are often other than they seem. Hence, in such cultures it is unlikely that indi-

viduals would have a need to repress aggression or resort to neurotic solutions, unless, of course, the aggressive fantasies were directed against the authorities. Under like circumstances, latent homosexuality may work itself out under the guise of comradeship and activity within the group. The need for a leader and father figure may also be met in certain groups by the passive submission to a dictator, who is clothed in the fantasy of omnipotence and omniscience. In passing, one may wonder what will be the ultimate effects upon civilization when such primitive instinctual demands are acted out, particularly since we are familiar with one type of individual in our own culture who employs this mechanism, the so-called psychopathic personality or instinctual character.

Various forms of communal activity—clubs, athletic groups—may be the medium for socialization of individuals. Here again the psychiatrist, often with the help of social workers, utilizes such resources in the community for psychotherapeutic purposes. Within these groups the individual has an opportunity for expressing at an accepted social level many of his needs—aggression in the form of competition and rivalry, for instance—and many of his libidinal trends in various sublimations.

Work, as such, particularly within our own culture, helps the individual to achieve many things—self-respect, independence, the capacity to help others, identification with the group, and attainment of new prestige values. The importance of work as an activity of sublimation is readily seen in the fact that so frequently inability to carry on work effectively is one of the first indications of a neurotic conflict; and the capacity for effective work may be the first indication of the solution of such a conflict.

Summary and Conclusion

It has been the writer's endeavor to sketch in somewhat summary fashion various dynamic factors which enter into different types of psychotherapy, as viewed from the perspective of psychoanalytic psychology. No intention is harbored of minimizing the value or efficacy of the other forms of psychotherapy. It would seem an obligation of the psychiatrist to evaluate the procedure he

employs, to attempt an etiological diagnosis of the problems he meets, and to plan such psychotherapeutic measures as will meet the individual needs of his patients. To do this, he must certainly bear in mind the difference between therapies aimed at symptoms and therapies aimed at causes. Under varying circumstances, varying techniques are needed. His aim should always be clearly in mind; and his procedure, insofar as possible, should be in the medical tradition, that is, with an awareness of the mechanism of the therapy employed.

82 Marlborough Street
Boston, Mass.

REFERENCES

1. Warren, Howard C.: Dictionary of Psychology. Houghton Mifflin Company, Boston, 1934. p. 218.
2. Hutchings, Richard H.: A Psychiatric Word Book. The State Hospitals Press, Utica, New York, 1935. p. 167.
3. Ellis, Sir W. C., M. D.: A Treatise on the Nature, Symptoms, Causes, and Treatment of Insanity, with Practical Observations on Lunatic Asylums, and a Description of the Pauper Lunatic Asylum for the County of Middlesex, at Hanwell, with a Detailed Account of Its Management. Samuel Holdsworth, Amen Corner, Paternoster Row, London, 1838. pp. 193-204.

PSYCHOTIC MANIFESTATIONS ASSOCIATED WITH PERNICIOUS ANEMIA*

BY FREDERICK J. DE NATALE, M. D.

Many excellent contributions have been made pertaining to nervous manifestations of pernicious anemia, but the psychotic features associated with this disease have not received the attention of medical men until recent years.

Addison,[1] in his original description of pernicious anemia, refers to the "feeling of languor, the indisposition to exercise and towards the end, the debility becomes extreme, the occasional wandering and indeed a terminal delirium, and at length expiration."

In 1900 Cabot[2] found, in 647 cases, 102, or 15 per cent, in which there were mental symptoms which he reported as follows: delirium 44, delusions 14, hallucinations 8, dementia 9, melancholia 3, and mania 3.

Pickett[3] in 1904, in his six cases of pernicious anemia, noted occurrence of confusion, with impairment of ideas of time and space, fabrications, delusions of persecution, hallucinations and occasional periods of excitement.

In 1905 Langdon[4] drew attention to a group of cases in which nervous and mental symptoms might precede a typical picture of pernicious anemia. He noted loss of inhibitions, peevishness and gradual mental deterioration. Camp,[5] in 1912, described a case of pernicious anemia with spinal cord changes, and a mental state resembling paresis; he felt that there was a similarity between the two diseases.

In 1913, Barrett[6] studied 11 cases and summed up the psychotic picture by saying that these patients have in common irritability and suspiciousness (which forms the groundwork for delusions of persecution), the content of which is usually influenced by the somatoneurologic findings. In over one-half of his cases there was a psychopathic family history.

*Read before the interhospital conference held at the New York State Psychiatric Institute and Hospital, New York, N. Y., April 18, 1940.

In 1919 Lurie[7] presented a study of four cases. He concluded that the psychoses may be classed with the symptomatic psychosis of a toxic organic nature, the whole delusional formation being vague, unsystematized, and loosely connected.

Jones and Raphael,[8] in 1920, reported a case with symptoms resembling those associated with arteriosclerotic psychosis, and emphasized difficulty at times to differentiate the two.

Darden and Hall,[9] in 1922, described a case of pernicious anemia with psychosis. They felt that there was too much confusion on the subject to arrive at any definite conclusion, but that there probably was some toxic state, a direct result of the pernicious anemia, causing mental symptoms.

Woltman,[10] in 1924, discussed the theories concerning the causation of mental symptoms. In Woltman's opinion the constitutional factor is of extreme importance; also he found some relationship between mild confusional states and the intensity of the anemia, not constant enough however to satisfy him that this knowledge was valid. In discussing the pathologic findings, he concludes, "Just what influence these changes have in the production of a psychosis, cannot be determined with certainty. They, no doubt, contribute to its development and yet they may be found in patients who have not had mental symptoms."

Hulett,[11] in 1928, emphasized the medicolegal importance of pernicious anemia with the report of a case showing a marked paranoid trend which resulted in the alteration of a will.

Richardson,[12] in 1929, reported the results of treatment in 67 cases of pernicious anemia at the Massachusetts General Hospital. In this group of 67, there were two cases of a true psychosis. Both had hallucinations and delusions of persecution.

Baker, Bordley, and Longcope,[13] in 1930, reported in a group of 44 cases of pernicious anemia that eight were found to have psychotic disturbances.

Phillips,[14] in 1931, stated that the mental symptoms may be merely a manifestation of character with irritability and changing mood, or that the mental disturbance may be more pronounced. The psychosis most frequently seen is of the paranoid type with delusions of persecution and suspicion, these delusions being more particularly directed against those who are responsible for the pa-

tient's welfare. The time of the onset of the mental disorder is variable; it may occur at any stage of the disease.

Young,[15] in 1932, analyzing 515 patients with pernicious anemia at the Peter Brent Brigham Hospital, found 4½ per cent of his cases showing psychotic traits. The mental symptoms varied considerably and included acute delirium, depression, paranoid states, memory and disposition changes.

A. W. Hackfield,[16] in 1932, wrote that in most cases where a psychosis was present, no definite etiologic relationship of the anemia to the psychosis was proven. Many of his patients presented histories of previous psychotic attacks and other predisposing factors. In the seven patients whom he treated he found that: (a) In most of the cases the psychosis developed following some environmental trauma. (b) Despite treatments the neurological signs progressed in several of the cases. (c) A history of suicidal tendencies or actual attempts on the part of the patient was obtained. Hackfield therefore contends that whatever changes may take place in the clinical course of pernicious anemia in no way tend to produce a parallelism in the course of the mental disorder; that pernicious anemia, *per se,* bears no direct etiologic relation to the production of the psychosis. In some instances the anemia acted as a precipitating or aggravating factor.

Wiltrakis,[17] in 1932, reported 10 cases of pernicious anemia at the Elgin State Hospital. Of these, four were diagnosed as psychosis associated with other somatic disease, pernicious anemia; two as manic-depressive psychosis, depressive type; two as paranoid dementia præcox; one as hebephrenic type of dementia præcox, and one as psychosis with cerebral arteriosclerosis.

Bowman,[18] in 1935, reported 23 cases of which 22 showed definite psychosis. Some of these illustrated how an early case of pernicious anemia may be regarded as a psychoneurosis with various psychological factors seen as responsible for the patient's condition; he stressed early examination of the blood. Concerning the type of mental picture shown in these 23 cases, it may be said that there were very few clearcut reaction-types. Eleven cases showed essentially the picture of organic confusion, three showed depression, two manic excitement, four showed a schizophrenic type of reaction, two a mixed picture of schizoaffective reaction, and one

showed a psychoneurosis. Bowman concluded from these 23 cases that there is no specific type of mental picture occurring with pernicious anemia.

Herman, Most and Jolliffe,[19] in 1937, reported that in a series of 255 patients with pernicious anemia, 40, or 15.7 per cent, showed mental symptoms of a degree to produce a psychosis. The mental picture of these psychoses did not show any character or diagnostic content. In this group of cases, in order of frequency, the mental reactions observed were acute confusional state, a paranoid condition, an affective reaction, and an organic deterioration.

Five cases observed at the Hudson River State Hospital are herewith reported showing mental symptoms associated with pernicious anemia. In each case the anemia was confirmed by laboratory examinations.

Case Reports

Case 1. M. S., said to be 39 years of age, was born in Russia. A brother is a patient in a mental hospital with a diagnosis of manic-depressive psychosis. M. S. was a normal, nine-month baby and, as far as is known, his early life and development were essentially negative. No history of illness was found. He came to the United States when about six years of age, received a common school education, and following this became a salesman, which line he followed until his admission to the State hospital.

He is described as being a fairly good mixer, had many friends of both sexes, was not shy, usually felt at ease in a crowd. He was easily discouraged and subject to emotional swings between elation and depression. Following the crash of 1929 he worried considerably because of the business depression.

It is stated that he felt perfectly well except that he was worried over business matters, until the beginning of 1932, when he began complaining that his eyes bothered him. He spoke of a burning sensation in his stomach, constipation, and indigestion. In July, 1932, he awakened from sleep in a cold sweat, to close the windows; this is the last he could remember until he found himself in the Troy Hospital, having been taken there for illuminating gas poisoning. The patient could find no reason for his action. This incident occurred on July 29, 1932. He remained in the Troy Hos-

pital until August 6, 1932, at which time he was discharged. Upon his return home he became depressed and thought that people were talking about him. Physically, he appeared weak, feeble, and accordingly was admitted to a sanatorium on January 20, 1933 where his mental picture was one of depression. He still believed that people talked about him. He expressed auditory hallucinations, thought that he heard people whispering to him, but was unable to understand what they were saying. He was admitted to the State hospital, February 22, 1933. On admission he appeared perplexed and apprehensive. There was some clouding of the sensorium. Physical examination showed a fairly well developed white male with coarse black hair; dry, scaly skin; spongy, bleeding gums; tonsils enlarged. An acute purulent conjunctivitis was noted; his pupils were equal, regular, and reacted promptly to light and accommodation. His gait was shuffling. His speech was slurred and tended toward stuttering. He manifested atrophy and weakness of all the muscles of the body, with diminished power in the lower extremities. His vibratory sense was diminished and his sense of position was disturbed. Blood examination revealed 2,300,000 red blood cells. A blood smear revealed the presence of microcytes, macrocytes, poikilocytes, shadow cells and nucleated cells. Hemoglobin was 70 per cent. Gastric analysis revealed a total absence of free hydrochloric acid. His blood pressure was 84/60. Blood Wassermann and spinal fluid examination were both negative. Blood sugar was 108 mgm. per 100 cc. of blood, and a diagnosis of pernicious anemia was made.

During the first month this patient was observed, he appeared confused, agitated, feeble, weak, unsteady, and was cared for in bed. He was defectively oriented for time, place and person. The first week in April he began to show some improvement. His sensorium appeared clearer, but he had an amnesia for the events of the preceding month. Under treatment his blood picture was restored to normal within several weeks. (He maintained an average count until about November of 1933.) He continued depressed, although this was not so marked as at the time of admission. However, in spite of energetic treatment, the patient began to complain of definite weakness in his extremities about the beginning of the year 1934. The man's condition became gradually worse, and

in spite of continued treatment, on June 6, 1934 his red blood count had fallen to 1,500,000 cells. Neurological signs on this date revealed a total absence of vibratory sensations up to the level of the umbilicus. Deep muscle sense was lost. On June 16, 1934, he lapsed into a delirious state from which he did not recover, and on June 26, 1934 the patient died.

Comment: In this case it is observed that for three years prior to admission the patient had been exhibiting emotional swings, characterized by depression and worry over financial affairs, which culminated in an unsuccessful suicidal attempt. In 1932, he began to express somatic complaints attributable to his gastrointestinal system, and coincidentally his depression became more pronounced so that at the time of his admission to this hospital in February, 1933, he appeared definitely depressed, apprehensive and perplexed. Shortly after his admission he exhibited a mental reaction simulating an organic reaction-type with confusion and clouding of the sensorium, from which he subsequently showed some improvement, only to lapse into a delirious state from which he did not recover. Unfortunately, permission for a postmortem examination could not be obtained.

Case 2. D. B. was born August 13, 1867. He is a native of New York State. No details were elicited concerning his early life and development. His wife was committed to this hospital in 1917 with a diagnosis of dementia præcox, paranoid type. As a young man he indulged freely in beer and stronger alcoholic beverages; he became intoxicated frequently, but during the last 14 years he claims to have been a total abstainer. He went through the rural schools and had the equivalent of two years of high school education. In his early life he spent his time between New York and Pennsylvania, but for 30 years had lived in Albany. He married in 1907 and stated that his married life had been congenial until 1917, when his wife became mentally ill and was committed to a mental hospital. She died on May 6, 1925, from an intestinal obstruction. He had worked on the railroad most of his life as a helper in construction gangs, but he had not been employed three years prior to his present admission. Since 1917 he had lived in rooming houses, and for the past three years had lived on charity.

At the age of 16, the patient suffered a fractured wrist. At the age of 18 he contracted a neisserian infection and received treatment from a physician. He was operated upon for the removal of a wen at the age of 54. When he was 65, he began to have considerable trouble with gas pains in his stomach, became unsteady on his feet, and complained of tingling sensations in his lower limbs.

The patient described himself as good-natured and pleasant; he stated that he had numerous friends of both sexes, and that he was never stubborn or suspicious. He admitted having been promiscuous sexually.

The patient denies that there is "anything wrong" with his mind but places the onset of his difficulties at about 18 months prior to his admission to this hospital. At that time he went to an old men's home, shortly after which his stomach began to trouble him. He experienced gas pains and he is convinced that his food was poisoned at the home. He stated that this continued throughout his residence there. The people seemed to be suspicious of him; they kept watching him. He remained there until the early part of the winter of 1933, when he left without permission, going to live in a home for the unemployed. He spent much time loitering about the railroad station and was warned by the police on several occasions to keep away. He noted that his stomach was again bothering him and had suspicions that his food was being poisoned wherever he ate. On the night of February 4, 1934, he felt severe pains in his abdomen and the next day he was sent to an observation hospital by the police. At that hospital, he informed the physician that he had fallen on the street on several occasions from weakness, and that his hands felt numb. He appeared restless, talkative, and still believed his food was being poisoned. He was admitted to this hospital on March 22, 1934. Here his mental picture was one of irritability and suspiciousness; he expressed the idea that poison was being put in his food. He exhibited memory defects both for recent and for past events, and he was defectively oriented for time. Physical examination showed numerous light brown pigmented areas on the skin. There was evidence of chronic myocarditis. His second sound was accentuated with a diastolic murmur at the apex. His blood vessels were sclerotic and his blood

pressure was 166/93. On neurological examination, it was noted that he walked with a wide base; his gait was unsteady and he swayed to both right and left. He was unable to write legibly, and in buttoning his clothes he required considerable time, the buttons often slipping from his grasp. His muscles showed wasting, and appeared flabby. Light touch was absent in both hands below the wrists and on the soles of the feet. Subjectively, he complained of dysesthesia such as tingling and numbness of the hands and feet, and cold sensations. His vibratory sense was absent from the umbilicus down. His sense of position appeared undisturbed. Laboratory examinations revealed a red blood count of 3,000,000, hemoglobin 60 per cent. Blood smear showed macrocytes, poikilocytes, anisocytosis and nucleated cells. Blood Wassermann was 4 plus. Spinal fluid was negative. There was one cell per cmm. and his globulin was normal. The patient was started on liver therapy, and although there was a marked improvement in his blood picture the mental reactions and neurological picture have remained essentially unchanged. He has continued to be suspicious, irritable, complaining that his food is being poisoned, and attributing his somatic complaints to this fact.

Comment: The patient's prepsychotic personality study is hardly conclusive, but it is apparent that he has shown some definite psychopathic traits. No distinct psychotic manifestations were noted until 1932 when he began to complain of somatic pains in the gastrointestinal region. He became suspicious, irritable, expressed ideas of reference, attributed his gastric complaints to the fact that poison was being placed in his food. Since his admission and up to the present writing, his mental picture has remained essentially unchanged except that he appears to have grown weaker. In this case, the patient's defects in memory and orientation may be explained on the basis of arteriosclerosis. However, he presents a typical paranoid reaction-type of mental disorder.

Case 3. A. R., is a white female. She was born on January 2, 1891 in New York State. Her early life is reported to have been uneventful. Her menses were established at the age of 13. They were normal up to one year prior to her admission to this hospital, and it is thought that she is now passing through the menopause. She attended the common schools and graduated from the eighth

grade at 14. She worked as a saleslady in various department stores, and kept up this employment until the time of her marriage. At the age of 41 she married a man five years her junior. She had known him for two years. Her married life was congenial for about three months, after which she became irritable, noisy, abusive, and after a year of married life her husband left her (about September, 1932).

Her personality is described as that of a somewhat seclusive individual. She almost never cared to go out, being content to remain at home. Her interests were limited: She did not enjoy dancing or any activity except reading at home.

This patient's medical history dates from 1929. At that time she complained of nausea and soreness of the tongue. She was seen by a physician, who on examination found a glossitis which he treated locally. Her symptoms, however, became aggravated and she complained of numbness and tingling sensations in the toes and fingers. In September, 1931, a blood examination revealed the hemoglobin to be 60 per cent. A differential smear showed polynuclears 54 per cent, myelocytes 2 per cent, hypochromia, poikilocytes and anisocytosis. In late September, 1931, she was again seen by the same physician. She complained of sore tongue and tingling sensations in the hands; the tongue showed atrophic changes. She was placed on liver therapy and from October, 1931, her blood picture rose from 2,477,000 erythrocytes with a 70 per cent hemoglobin to 5,211,000 erythrocytes and a 90 per cent hemoglobin in February, 1932. However, from this point on, she did not follow the physician's instructions: Neglecting to take treatments, from May of 1932 to January of 1933 she became increasingly ill. She complained of indigestion, vomiting, numbness and tingling sensations.

Coincidental with these physical complaints, she was experiencing difficulty at home with her husband. She began to develop the idea that people in the neighborhood were talking about her, that her husband was putting poison in her food. She became suspicious of everyone about her. Finally her mental condition became so aggravated that on January 9, 1933 she was admitted to the Hudson River State Hospital.

Physical examination on admission showed a white female weighing 102 pounds. Her heart and lungs were essentially negative. She complained of headache in the vertex and in the occipital region. For the past three years, she said, she had suffered dizziness, and weakness in the legs. Neurological examination indicated that her pupils were equal, regular, and reacted promptly to light and accommodation. There was weakness of all movements, more marked in the lower extremities. She was unable to walk or stand on her feet without support. Her knee jerks were hyperactive and there was a bilateral positive Babinski reflex. Light touch was absent on the soles of the feet. Her sense of position was disturbed. Vibratory sensation was lost in the lower extremities. Sensation was disturbed from the level of the second lumbar vertebra down. Pain sensation appeared to be delayed. Laboratory examinations revealed a red blood count of 1,900,000, hemoglobin 60 per cent. Blood smear revealed the presence of nucleated cells, poikilocytes, microcytes, and macrocytes. Gastric analysis revealed a total absence of free hydrochloric acid, with a combined acidity of 12 per cent.

The patient was immediately placed on liver therapy, but showed negligible improvement in the neurologic and mental spheres. Her ideas of persecution became more marked. She began to express the thought that her bed and the chairs were wired with electricity, and expressed a marked persecutory trend directed against her husband. She felt that poison was being put in her food and that the nurses about her were spies attempting to find out what she was doing. Since her admission and until the present writing, the patient's mental picture has remained essentially unchanged.

Comment: This case presents a medical history of pernicious anemia dating from 1929. The patient's prepsychotic personality may be definitely labeled as of schizoid type. Her psychosexual development did not follow a normal course. Although physical symptoms appeared in 1929, no psychotic manifestations were noted until about three months after her marriage in 1931, when she began to develop a schizoid mode of reaction. The stress of her marital readjustment, combined with her physical illness, in all probability lowered her psychic threshold and she developed a definite projection mechanism directed against her husband. Since

her admission and up to the present writing, she presents a typical paranoid schizophrenia with delusions of persecution (including ideas of being poisoned) and auditory hallucinations.

Case 4. W. S. T. was born in New York City, December 28, 1874, and was 59 years of age at the time of his admission. One maternal cousin is said to have died in a mental hospital in 1929 and the patient's wife was, at the time of his admission to this hospital, a patient in a mental hospital with a diagnosis of manic-depressive psychosis, depressive type. Nothing abnormal was noted during the present patient's early life and development. He was graduated from a university in 1898, and he went into the contracting business. He had made a success of his work until 1930, when during the economic depression he lost most of what he had earned. He has had numerous injuries of a minor nature. Four years ago he suffered a fractured hip, and during the last eight years has had recurrent attacks of malaria. He had been a moderate user of alcohol until one year prior to his admission, when he began to indulge excessively.

The patient is described as always having been a careful, practical, deliberate individual with a wide range of interests. He was considered bashful, but easy to get along with; he was easily depressed by his failures. The precipitating causes of his psychosis may be listed as his own physical illness, the commitment of his wife in 1929 to a mental hospital, and the economic depression.

The patient became somewhat depressed after his wife's illness; from 1929 until 1931 he worried over financial matters. In the summer of 1931 he went to Europe with his daughter, but on his return his appetite became poor. He complained of indigestion, became morbid, and at this time was informed by a physician that he had anemia. Having been advised to take a cruise, in January, 1932, he started on a trip through the Mediterranean. During the excursion he experienced two emotional upsets, in one of which he wounded the steward, and was placed in a straightjacket aboard ship. On his arrival in New York, in March, 1932, he developed the idea that the shipping company had hired nurses to watch him. He thought that the Roman Catholics were persecuting him. He thought that there were dictaphones in his room to pick up everything he said. He was seen by a New York specialist and was given injections, but

he became unmanageable and thought poison was being injected into his body. In April, 1932, he was admitted to a private sanatorium, where he remained until February, 1934. While under care there, he had periods of emotional instability, felt that his bodily actions were being controlled by outside influences. He also heard voices of men and women commenting on his thoughts. He was admitted to this hospital on February 3, 1934. On initial physical examination, his heart and lungs were found to be essentially negative. He showed a kyphosis of the dorsal spine with some evidence of peripheral sclerosis. His blood pressure was 170/84. In the neurological sphere, he complained subjectively of tingling and numbness in his hands and feet. There was no atrophy of the muscles. There was some diminution of the vibratory sense from the left iliac spine down. Deep muscle sensibility was intact. Laboratory examination revealed his red blood count to be 2,400,000 with a hemoglobin of 61 per cent. His smear showed microcytes, macrocytes and poikilocytes. Gastric analysis revealed a total absence of free hydrochloric acid with a combined acidity of 8 per cent. He was immediately started on liver therapy. Following the administration of liver, his blood picture improved. However, his mental reactions remained essentially unchanged. He continued to express ideas that he was being persecuted by the Catholics, that machines had been placed in various parts of the ward which could read his mind, and that his thoughts were transmitted to persons outside. He experienced auditory hallucinations, believing that people were talking about him and were accusing him of doing wrongful things. His condition has remained essentially unchanged.

Comment: It is known from the patient's personality study that he was easily depressed by failures; in particular, he had suffered a mental breakdown following the crash in 1929, when he lost all his savings. This fact, combined with the commitment of his wife to a mental hospital and worry over his own physical illness, seems to have helped in bringing on his present mental disorder. He first exhibited mental symptoms in the form of two episodes of emotional instability which finally passed over into a definite schizophrenic reaction-type of psychosis. This patient died on January 23, 1938, the immediate cause of his death being lobar pneumonia.

Case 5. M. R. was born March 20, 1875. There is no information relative to her early life and development. She is said to have never had any serious illnesses or operations. Little is known of her menstrual history other than that it is definitely stated that she has passed her menopause. She came to the United States about 20 years ago. She had married at the age of 20 and her husband was 20 years her senior. He died about 10 years after they were married. There was one child by this union. On arrival in this country, she worked in a laundry and then for about five years conducted a boarding house. She has been idle for the past 11 years, living with her daughter.

This patient is described as always having been unpleasant, stubborn, domineering, impulsive, quick-tempered, and extremely selfish. She rarely took the advice of anyone. After her daughter's marriage she continued to manage the daughter's affairs; when crossed, she would fly into a rage.

The information given by the patient's daughter regarding the patient's illness stated that, to her, her mother had never appeared normal: She had always had a "peculiar" personality and a mean disposition. In about 1932 she was told by a physician that she had pernicious anemia. She soon became greatly concerned about herself, going from one physician to another, never satisfied. She went from hospital to hospital, in each case leaving before any sort of treatment could be instituted. She would fly into rages at her daughter's home, and in general showed marked emotional instability. She was urged to seek voluntary admission to a mental hospital, which she did on March 15, 1934. On initial physical examination, she appeared a thin, undernourished female, complaining of right-sided pain in the chest and abdomen, and of diarrhea. Her heart and lungs were negative on examination. She complained of tingling sensations in her fingers and toes. Her pupils were equal, regular, and reacted promptly to light and accommodation. Her gait, muscular coordination and balancing were good. The sensory examination was essentially negative. Laboratory examination revealed a red blood count of 2,600,000; hemoglobin was 60 per cent. The blood smear revealed the red cells to be distorted.

Gastric analysis showed a total absence of free hydrochloric acid and a combined acidity of 6 per cent. Immediately following her admission she was placed on liver therapy.

The patient remained in the hospital for several years. During this period, her mental illness was characterized by frequent outbursts of emotional instability during which she would become noisy, excitable, disturbed, accusing various individuals about her of mistreating her. These periods would subside and she would be comfortable for several weeks, following which she would repeat the episodes of emotional instability.

Comment: This patient's prepsychotic personality can be definitely classified as that of a psychopath. In 1932 when informed that she was suffering from pernicious anemia, her psychopathic traits became more accentuated and she became a definite problem with respect to care at home. Her periods of emotional instability became more pronounced and finally she sought admission as a voluntary patient. While under care at this hospital, her mental reaction has been characterized by excitement, irritability and suspiciousness.

Observations

In analyzing the group of cases presented it can be definitely stated that there does not appear to be any well-defined mental syndrome characteristic of mental disorders associated with pernicious anemia. The mental symptoms vary from organic reaction-types of mental disorder to well-defined types of schizophrenia. Precipitating causes in addition to the presence of pernicious anemia are commonly noted: In the cases discussed here, a wife's mental illness and worry associated with financial stress were observed. The prepsychotic personality is also worthy of study: One patient in this group (case 1) was easily discouraged and subject to emotional swings of elation and depression. In case 2, definite psychopathic traits were noted such as vagabondage and sexual promiscuity. Case 3 represents a well-defined introvert type of personality makeup, characterized by seclusiveness and limited

interests; and in case 4 it is again noted that the patient was easily depressed by his failures. The last case, No. 5, was that of a definitely psychopathic individual who was stubborn, selfish, quick-tempered, and whose mental symptoms were merely an accentuation of her usual personality makeup. Certain symptom-complexes, however, appear to be more frequently met, such as irritability, suspiciousness, ideas of reference and well-defined paranoid formulations, although they cannot be stated to be characteristic of the disease. The character of mental symptoms seen in the patient may vary from time to time during the course of illness. In case 1, the psychosis began as a well-defined depression, developing into an organic reaction-type, followed by a definite paranoid reaction and terminating in a delirium.

Conclusions

1. No specific type of mental disorder is found to be associated with pernicious anemia.

2. Sufficient causes are generally found, either psychogenic or external in nature, which in themselves would be sufficient to produce a mental illness.

3. Pernicious anemia, *per se,* may be considered only as a stimulating or aggravating factor in the production of the psychosis.

4. The prognosis of the mental disorder associated with pernicious anemia does not appear to be different from the prognosis of similar reaction-types met without evidence of physical disease.

5. The onset of the mental symptoms does not appear to bear any definite relation to the physical disorder. Symptoms may appear either before, at the onset, or during the course of pernicious anemia.

6. Careful study of the personality makeup will often reveal outstanding personality defects.

7. Symptom complexes characterized by irritability, suspiciousness, ideas of reference and well-defined paranoid formulations, appear to be most frequently encountered.

Hudson River State Hospital,
Poughkeepsie, N. Y.

BIBLIOGRAPHY

1. Addison: Anemia—Disease of the suprarenal capsules. London Med. Gaz., 43:517, 1848.
2. Cabot (quoted by Rogers, A. A.): Disturbances of the central nervous system, accompanying pernicious anemia. Jour. Nerv. and Ment. Dis., 62:693-699, 1915.
3. Pickett, W.: Mental symptoms associated with pernicious anemia. Am. Jour. Med. Sci., CXXVII: 1032-1936, 1904.
4. Langdon, F. W.: Nervous and mental manifestations of pre-pernicious anemia. J. A. M. A., XLV:1635, 1638; 1905.
5. Camp, C. C: Pernicious anemia causing spinal cord changes and a mental state resembling paresis. Med. Rec., LXXXI:156-158, 1912.
6. Barrett, A. M.: Mental disorders and cerebral lesions associated with pernicious anemia. Am. Jour. Insan., LXIX:1063, 1913.
7. Lurie, L. A.: Pernicious anemia with mental symptoms. Arch. Neurol. and Psychiat., 4:55, July, 1920.
8. Jones, B. L., and Raphael, T.: Differential diagnostic difficulties in pernicious anemia with associated mental disorder. Arch. Neurol. and Psychiat., 4:55, July, 1920.
9. Darden, O. B., and Hall, J. K.: The psychosis associated with pernicious anemia. South. Med. and Surg. Jour., 84:11, November, 1922.
10. Woltman, H W.: The mental changes associated with pernicious anemia. Am. Jour. Psychiat., 3:435, January, 1924.
11. Hulett, A. G.: The psychologic and medico-legal aspects of pernicious anemia. Med. Jour. and Rec., 127:1, January, 1928.
12. Richardson, W.: Pernicious anemia. N. E. Jour. Med., March, 1929.
13. Baker, B. M., Jr., Bordley, J., III, and Longcope, W. T.: The effects of liver, and liver extract upon the symptoms and signs referable to the nervous system in pernicious anemia. N. E. Jour. Med., 13:817, November, 1930.
14. Phillips, N. R.: Mental disorders associated with pernicious anemia. Jour. Ment. Sci., 77: July, 1931.
15. Young, R. H.: Neurologic features of pernicious anemia. J. A. M. A., 99:612-614, August, 1932.
16. Hackfield, A. W.: Studies of etiologic relationship, critical review with case reports. Jour. Nerv. and Ment. Dis., 76:31-48, July, 1932.
17. Wiltrakis, G. A.: Psychosis with pernicious anemia. Collected and contributed papers of Elgin State Hospital, December, 1932.
18. Bowman, Karl M.: Psychosis with pernicious anemia. Am. Jour. Psychiat., 92:2, September, 1935.
19. Herman, M., Most, Harry, and Jolliffe, Norman: Psychosis associated with pernicious anemia. Arch. Neurol. and Psychiat., 38:2, August, 1937.

THE USE OF CURARE IN MODIFYING METRAZOL THERAPY

BY R. W. GRAY, M. D., F. L. SPRADLING, M. D., AND A. H. FECHNER, M. D.

Metrazol has been used during the past few years as an agent to produce convulsions in the shock therapy of schizophrenia, involutional melancholia, and other psychoses. In this connection it has been widely accepted as a drug of value. The end results of its use as reported in a large number of cases have been satisfactory except for a variety of injuries caused by the severe convulsions. Persistent reports have been made enumerating fractures of the femur, pelvis, and dorsal vertebrae. Dislocations of the shoulder and mandible, and muscle and tendon strains have been frequently produced.[1-8]

The need for modification of metrazol convulsions has, therefore, been of interest to many, and much preliminary study and investigation have resulted. At the Lincoln State Hospital, Lincoln, Neb., the authors have resorted to many postures, supports, splints, bandages, and restraints in an unsuccessful effort to prevent accidents. Spinal anesthesia proved of some value but was discontinued because of the undesirability of frequent administrations and because of its failure to control convulsions in the upper part of the body.

Experimental work on the dog has shown the efficacy of curare in modifying muscle contractility over the entire body. The drug produces a paralysis of skeletal motor muscles. It has a selective depressive action on motor nerve endings and altogether excludes sensory endings. The results of its action are first seen in the short muscles of the toes, eyes, and ears, then in arms and legs, followed by the head and neck. Finally, the thoracic, diaphragmatic, and abdominal respiratory muscles are affected in order. The value of curare in modifying the metrazol convulsion was first described by Bennett.[9]

The production of a stable and well-standardized preparation of curare has made it easily possible to graduate the degree of metrazol convulsion in the human subject. The preparation is an aqueous solution, each cc. of which represents a potency equivalent to 10 mgm. of the crude drug. It is given in a dosage of 1 cc. to each

20 pounds of body weight. Within one to one and one-half minutes after administration a ptosis of the eyelids is seen, followed quickly by inability to lift the hands and feet easily from the bed. The head is also lifted with difficulty. The degree of these manifestations is variable and is not considered of particular value in measuring the efficacy of the curare. Dosage of curare, speed of injection, and amount of time intervening between the administration of the curare and metrazol are all factors which are of more importance.

The mode of administration has been determined by experience and is simple. The fasting patient is prepared as for all metrazol treatments which are usually given three times a week. The curare is injected intravenously within 30 to 60 seconds elapsed time. After injection the needle is allowed to remain in the vein while the syringe is removed and a second syringe containing the metrazol is attached in its place. Three minutes after the completion of the curare administration, the metrazol is injected in the usual rapid manner. A much modified but otherwise typical metrazol convulsion immediately ensues. The usual clonic, tonic, clonic sequence is observed. Moderate cyanosis followed by pallor is present. During the tonic spasm the degree of rigidity is reduced to the point where arms and legs may be easily moved about by the attendant. The intense and violent clonic spasm of the unmodified metrazol convulsion is absent. In its place is a much more gentle action which in force is incapable of severely straining any body structure. The legs are not raised from the bed, nor is opisthotonos observed. It is not necessary to protect the tongue with mouth gag or tongue blade. Rarely is the patient incontinent nor does ejaculation occur. Little or no disturbance of blood pressure is seen. The usual postconvulsive excitement is greatly modified or absent.

In the series of cases here discussed, respiratory depression has been of sufficient severity in but three cases to necessitate special treatment. It was readily controlled by the intravenous injection of one to two cc. of 1:2000 prostigmin methylsulphate. In these

cases artificial respiration was also used for from three to five minutes. Respiration was quickly reestablished as the curare action proved to be fleeting. No circulatory depression has been evident in any case.

One group of patients was treated with metrazol alone and later with metrazol modified with curare. This group has not been enumerated in the results here reported. A few of these cases have had fractures while taking unmodified metrazol treatment. Because of the curare modification, it has later been possible to continue their satisfactory treatment without further fractures or involvement of old injuries. Many patients have received treatment, who, because of general feebleness or advanced age, would otherwise have been refused shock therapy.

Results

Fifty patients have been treated with the combination of curare and metrazol. All were carefully examined to rule out the possibility of previous trauma or disease. Seventeen of them were X-rayed with this purpose in mind. None of them had been previously treated with metrazol shock therapy, nor had any of them been subject to convulsive disorders. They represented both sexes, with ages ranging from 16 to 70. Four hundred and eighty-five convulsions have been produced with an average of 9.7 per patient. After completion of treatment each patient has been carefully and painstakingly examined for evidence of vertebral fracture or injury to the intervertebral disc, with a complete lack of positive findings. Those cases in which there has been the least doubt of a spinal trauma have been X-rayed. Twenty-four such cases have been checked with right lateral oblique and flat plate Roentgenograms. Evidence of trauma has been lacking in each instance. There have been fractures of no other bones. No patient has complained of soreness or has given any other evidence of strain of soft parts. Improvement following treatments has been in all respects as satisfactory as the results following 1,582 convulsions produced in this hospital by unmodified metrazol.

Conclusions

1. Metrazol modified by curare has been safely given to 50 patients ranging in age from 16 to 70. No resulting trauma of any description has been demonstrable.

2. It is possible with this method to treat patients who, because of debility, age, or disease, might not be safely treated otherwise.

3. The satisfactory results formerly attributed to unmodified metrazol are still present and apparently not changed by the addition of curare.

Acknowledgment

A preparation of curare under the trade mark "Intocostrin" was supplied for experimental purposes by E. R. Squibb & Sons, New York City.

Lincoln State Hospital
Lincoln, Nebr.

REFERENCES

1. Polatin, P., Friedman, M. M., and Horwitz, W. A.: Vertebral fractures produced by metrazol induced convulsions. J. A. M. A., 112:1684, June 3, 1939.
2. Bennett, B. F., Jr., and Fitzpatrick, C. P.: Fractures of the spine complicating metrazol therapy. J. A. M. A., 112-2240, June 3, 1939.
3. Hamsa, W. R., and Bennett, A. E.: Traumatic complications of convulsive shock therapy. J. A. M. A., 112:2244, June 3, 1939.
4. Dick, A., and McAdam, W.: Cardiac complications in cardiazol treatments; observations in four cases. Jour. Ment. Sci., 84:999, November, 1938.
5. Bennett, A. E.: Convulsive shock therapy in depressive psychoses. Am. Jour. Med. Sci., 196:420, September, 1938.
6. Graves, C. C., and Pignataro, T. P.: Injuries sustained during the course of metrazol shock therapy. Psychiat. Quart., 14, January, 1940.
7. Furst, W.: The force required to crush vertebrae: its probable mechanical relation to the post-metrazol fracture. Psychiat. Quart., 14, p. 477, July, 1940.
8. Robinson, G. W., Jr., and Shelton, P.: Metrazol therapy in the face of severe physical disorders. Psychiat. Quart., 14, p. 388, April, 1940.
9. Bennett, A. E.: Preventing traumatic complications in convulsive shock therapy by curare. J. A. M. A., 114:322, January 27, 1940.

THE PSYCHOPHYSIOLOGICAL ACTION OF BETA-ERYTHROIDIN HYDROCHLORIDE

BY SAMUEL R. ROSEN, M. D., AND MORRIS V. BORENSTEIN, M. D.

The recent development of the therapeutic use of beta-erythroidin hydrochloride, especially in the psychiatric field, has stimulated considerable interest in the mode of action of this drug. The writers have had the opportunity to observe its action in 60 psychiatric cases receiving a total of 250 treatments in which beta-erythroidin hydrochloride was used to modify the severity of the therapeutic metrazol convulsion.[1, 2]

Historically, the crude form of the drug has been known for many years as one of a number of derivatives of the genus *Erythrina* which possesses a curare-like action.[3] Although never employed as an arrow poison, the plant juices are said to have been used by South American Indians in paralyzing fish, thus rendering their capture much easier. The drug was first used therapeutically by Burman[4] who employed it as a substitute for curare in the treatment of spastic dystonia. Erythroidin had been previously prepared and studied by Folkers, Koniuszy, Major and Unna.[5-8] They demonstrated a definite paralytic action in animals. Burman felt that this paralytic action was markedly inferior to that obtained with curare and pointed out that relatively much larger doses of erythroidin were needed. In a later paper[9] he summarized his findings concerning the action of the drug. He used doses, however, which, in the experience of the writers, are inadequate to produce the complete paralytic action required.

[The present form of the drug beta-erythroidin hydrochloride, is a purified isomer from the erythroidin-hydrochloride, which was derived from the seeds of *Erythrina americana* Mill.* and which was used by Burman. The present form was first studied in dogs by Ziegler and Cominole with one of the present writers (S. R.)[1] for its modifying action on the severity of the metrazol convulsion.] This experiment was undertaken as a result of the large number of fractures due to metrazol therapy reported in

*Supplies of the drug were prepared and obtained through the cooperation of Merck and Company, Rahway, N. J.

recent literature. It was hoped that metrazol treatment would be continued if some means could be found to reduce the force of the muscular contractions producing the vertebral and femoral fractures. In dogs, complete motor paralysis was obtained with a dose of 4 mgm. per kg. weight of dog. There were no concomitant effects. Recovery was spontaneous within 15 to 30 minutes. Severe respiratory symptoms appeared with doses of 6 mgm. per kg. Predetermined convulsant doses of metrazol when given to the "erythroidinized" animal produced very mild seizures with spontaneous recovery in 15 to 20 minutes.

Encouraged by these results, Cameron, Ziegler and one of the present writers (S. R.)[2] applied the method to a series of schizophrenic and manic-depressive patients. The action of the beta-erythroidin hydrochloride was strikingly altered in human beings. Complete motor paralysis was never obtained, while on the other hand, the action of the drug could be readily observed in a complex of signs and symptoms falling into several categories, namely cerebral, ocular, pupillary, facial, articulatory, pharyngeal, laryngeal, circulatory, respiratory and finally, general neuromuscular reactions.

The drug is prepared in sterile 10 per cent aqueous solution by vacuum filtration and is gradually injected by the intravenous route at the rate of 200 mgm. per minute. (In subsequent treatments this may be speeded up to 400 mgm. per minute.) The cerebral symptoms are usually the first to appear. Within 30 to 40 seconds the patient may say that his head feels funny," that he feels "light in the head," or that he thinks he is "swimming away." Occasionally some apprehension may appear, especially in cases where the patient's lack of insight does not permit his comprehension of the routine pretreatment explanations of what he may experience during the treatment. This initial action may pass off as the injection is continued, to return later in varied forms on the same central theme as other symptoms make their appearance.

Ptosis next develops usually after the fourth and almost always before the eighth minute of injection (between 800 and 1,600 mgm.). The patient may begin with "my eyes feel heavy" after 300 or 400 mgm., a gradual bilateral but occasionally unequal ptosis soon following. Sudden onset of complete ptosis during the space

interval of 200 mgm. injection indicates idiosyncrasy, which will be discussed at another point.

The facial symptomatology is the least definite. There is a gradual change in the facies to the myasthenic type, in which ironing-out of muscular folds and some degree of mandibular drooping usually appears by the seventh or eighth minute. This varies widely, however. Little or no salivary drooling has been observed. Some lacrimation has been occasionally noted.

Dysarthria is generally a fairly late symptom, but frequently the patient may state after the fourth or fifth minute that he "can't talk" although his articulation may be reasonably distinct. This is closely associated with pharyngeal and laryngeal involvement, although the latter does not become severe until a very late stage. Nasal voice appears over a range varying from the seventh to the tenth minute and is then rapidly followed by dysarthria with final onset of anarthria, a point which definitely marks a terminal phase of the action of the drug. Rapid development of dysarthria points to idiosyncrasy and added caution in further administration.

Circulatory effects tend to be mild in the average case. The blood pressure may show a systolic drop of 10 to 30 mm. of mercury associated with a much slighter diastolic dip, subject, however, to frequent vacillation with the patient's emotional state. The pulse rate may fall somewhat, but this is not a regular occurrence. Sudden fall in blood pressure, or rarely a marked and sustained elevation, indicates idiosyncrasy. The blood pressure drop seems to be due to overrapid administration of the drug. Upon decreasing the rate of injection of the drug, the blood pressure may rise again while the paralytic symptoms remain unchanged. Cases with a definite alcoholic history have tended to exhibit these changes most frequently.

The respiration may quiet somewhat during the first six or seven minutes of injection, after which the patient may suddenly state that he "can't breathe" and may develop hyperpnea. This frequently has an emotional basis and may subside. Again, it may go on to real respiratory embarrassment associated with gasping and beginning cyanosis. This indicates a terminal stage in the injection and demands immediate interference, as will be later discussed. The respiratory picture is, peculiarly enough, closely as-

sociated with the amount of laryngeal involvement which has occurred, inasmuch as laryngoscopic examinations made at the time of maximum respiratory embarrassment reveal a marked degree of narrowing of the airway due to relaxation of the laryngeal musculature.

The pupillary signs are important in that they can be used as the therapeutic end-point of administration of the drug, provided that none of the other symptoms have attained terminal values previously. Before beginning injection, the size of the pupils should be carefully noted and the speed of reaction to light observed. Then usually at about the time that early dysarthria appears, the pupils begin to dilate, anisocoria may occur, either or both pupils becoming somewhat eccentric in shape. This usually occurs in a time-injection range varying from the sixth to the tenth or twelfth minute. The reaction to light becomes sluggish, and within another minute or less the pupils become fixed. This point is usually the stage past the therapeutic end-point, which should be considered as that point marked by moderate pupillary dilatation and moderate sluggishness in the reaction to light. It is usually at this phase that the metrazol is injected.

The last sign-symptom complex is a rather broad one, the neuromuscular group. As the injection progresses to the fifth or sixth minute, occasional jerkings of the various extremities may be observed. The deep tendon reflexes become more active. Equivocal Babinski reflexes have been noted, although their duration was fleeting. Gradual and general muscular weakness sets in after the seventh or eighth minute, but complete muscular paralysis has not as yet been encountered. The patient may be able to stand on an unsteady base, but totters and falls on attempting to walk. The uplifted upper extremities soon sink to the level of the bed. No grasping reflex or cogwheel rigidities have been observed. In general the more muscular the patient, the later the neuromuscular and other signs and symptoms appear in the course of the administration of the drug. The neuromuscular effect is markedly emphasized when metrazol is given, and it is then possible to contrast the usual spasticity in the pure metrazol convulsion with the flaccidity of the extremities in the "erythroidinized" patient.

Foregoing, is a composite exposition of the action of beta-erythroidin hydrochloride in a typical patient. The various signs and symptoms have been discussed in the order in which they most frequently have appeared. However, any one or several of these may be absent or present to only a slight degree. The cerebral, ocular, articulatory and the pupillary manifestations are fairly constant, reliable indices of the increasing degree of "erythroidinization" which has been attained. Circulatory and respiratory symptoms may in general be included in the realm of idiosyncrasy.

Idiosyncrasy to this drug falls into two degrees. The lesser degree is usually noted by sudden development of full ptosis or dysarthria, along with some mild respiratory distress and perhaps a sharp drop of 25 mm. of mercury in systolic blood pressure. Under these circumstances prostigmin, the specific antidote, should be given intravenously, usually 2 to 4 cc. of a 1:2000 solution, and the convulsant metrazol dosage should immediately follow. The modified seizure occurs and, by the time the last clonic twitch is over, the prostigmin has taken effect and erythroidinization is largely overcome. In the more serious degree of idiosyncrasy, the circulatory and respiratory symptoms develop rapidly, usually within the fourth to sixth minute of injection (800 to 1200 mgm.). The pressure falls rapidly, pulse becomes indistinct, cyanosis and gasping are marked. Prostigmin (2 to 4 cc.) should be given intravenously and artificial respiration applied. Metrazol should not be given, and the treatment is left incomplete. If necessary, a total of 6 to 8 cc. of prostigmin may be safely given, 2 cc. at a time, in such emergencies without any worse after-effect than somewhat prolonged borborygmi. Occasionally some nausea and vomiting may occur. This is readily counteracted with atropine 1/120 gr. subcutaneously. No fatalities have occurred in five instances in which the above sequence of events took place. Furthermore, the drug in every instance was safely given to each of these patients several days later and the treatments satisfactorily completed by reducing the rate of administration to 100 mgm. per minute. The idiosyncrasy then seems to be directly related to the speed of injection.

Paradoxically, the total dosage bears little or no relation to the patient's weight. However, the degree of muscular development

seems to be the determining factor, the more muscular patients requiring larger doses to attain the therapeutic end-point. Dosage varies from 800 mgm. to 2400 mgm., although the main run of treatments required from 1400 to 1800 mgm. for satisfactory modification of the metrazol seizure. The drug, which has now been shown to be a tertiary ammonium base, acts upon the motor end-plate inactivating the acetyl choline mechanism by releasing choline esterase, thus inhibiting transmission of the motor impulse to the muscle bundle. The prostigmin overcomes the erythroidin action by its inhibition of choline esterase activity; thus the acetyl choline may continue to facilitate transmission of the motor impulse at the myoneural junction.

The use of the beta-erythroidin hydrochloride does not necessitate larger doses of metrazol to produce a satisfactory seizure, doses between 4 and 8 cc. of metrazol having been found quite adequate in the great majority of cases. Nor is there any evidence of day-to-day accumulative action of the beta-erythroidin hydrochloride. Once the dosage is determined for a given patient, the end-point will not vary by more than 200 mgm. All effects of the drug are spontaneously relieved within one hour or can be removed with prostigmin within one to two minutes following intravenous injection.

Cameron found that with doses as high as 800 mgm. only drowsiness was obtained. The degree to which this symptom appeared varied considerably among different patients. It was noted that the effect lasted three to five hours in most cases. This finding is essentially similar to that which Burman[9] reported when he accidentally swallowed about 100 mgm. of the drug in orange juice.

Summary

The psychophysiological action of beta-erythroidin hydrochloride has been presented together with a brief survey of the development of the drug and its therapeutic utilization in the modification of the metrazol convulsion. The problem of idiosyncrasy to this drug has been discussed and the use of prostigmin as an antidote described. The writers wish to stress the fact that the use of this drug, with the above facts well in mind, can be considered a safe procedure.

Acknowledgment

The writers are indebted to Dr. D. Ewen Cameron of the department of neuropsychiatry of the Albany Hospital for his data in connection with the results of oral administration of beta-erythroidin hydrochloride.

Department of Neuropsychiatry
Albany Hospital and Albany Medical College
Union University
Albany, N. Y.

REFERENCES

1. Rosen, S. R., Ziegler, J. B., and Cominole, B.: Pharmacologic modification of the metrazol convulsion. Jour. Am. Pharm. Ass., 29:164, April, 1940.
2. Rosen, S. R., Cameron, D. E., and Ziegler, J. B.: The prevention of metrazol fractures by beta-erythroidin hydrochloride. Psychiat. Quart., 14:3, July, 1940.
3. Krukoff, B. A.: The American species of Erythrina. Brittonia, 3:205, October, 1939.
4. Burman, M. S.: Therapeutic use of curare and erythroidine hydrochloride for spastic and dystonic states. Arch. Neur. and Psychiat., 41:307, February, 1939.
5. Folkers, K., and Major, R. T.: Isolation of erythroidine, an alkaloid of curare action, from Erythrina Americana Mill. Jour. Am. Chem. Soc., 59:1580, 1937.
6. Folkers, K., and Unna, K.: Erythrina alkaloids. II. A review and new data in the alkaloids of species of the genus Erythrina. Jour. Am. Pharm. Ass., 27:693, 1938.
7. Folkers, K., and Koniuszy, F.: Erythrina alkaloids. III. Isolation and characterization of a new alkaloid, Erythramine. Jour. Am. Chem. Soc., 61:1232, 1939.
8. Folkers, K., and Unna, K.: Erythrina alkaloids. V. Comparative curare-like potencies of species of the genus Erythrina. Jour. Am. Pharm. Ass., 28: (in press), 1939.
9. Burman, M. S.: Clinical experiences with some curare preparations and curare substitutes. Jour. Pharm. and Exper. Therap., 69:143, June, 1940.

PARANOID AND COMPULSIVE SYMPTOMS ASSOCIATED WITH OCULOGYRIC CRISES

Report of Two Cases

BY MORRIS W. BRODY, M. D., AND HERBERT FREED, M. D.

Mental symptoms, particularly obsessions and compulsions, have been commonly observed in postencephalitic states. Many reports in the literature tend to correlate compulsive manifestations with oculogyric crises.[1] These observations are not sufficiently conclusive and are open for further consideration and study. Having observed two such cases simultaneously, the writers are presenting them for discussion.

Case Reports

Case 1. This patient was a 36-year-old married, white female. One sister was in a mental hospital, and a maternal aunt was said to be "insane." A nephew was epileptic.

The patient, born in Russia, saw much bloodshed as a result of local pogroms, and during childhood had fears of being raped or "sold into white slavery" (common talk of the people). At the age of 14 she witnessed her nephew having an epileptic convulsion. This frightened her, and she thinks that on such occasions he experiences the same trouble with his eyes as that which she is now having. At the age of 15 the patient immigrated to this country. She was described as friendly, outgoing and well liked. She made a satisfactory marital and sexual adjustment.

The onset of her present illness, in 1934, was sudden and dramatic. A man gained entrance to her house and attempted to violate her. He was frightened away and the patient was not seriously hurt. Her first thought was, "I am so disappointed in America." The following night her husband noted that something was "wrong" with the patient after she went to a nearby drugstore, saying someone was going to rob it and accusing her husband. She became afraid of crowds, developed vertigo, and gave up all social activities. She felt that her husband was to be

blamed for a crime, that her children would be harmed, that people were laughing at her because she was poor. She had recurring dreams in which persons committed crimes of violence.

In July, 1934, she attended the Temple University Neurologic Clinic, where neurologic examination was negative and no mention was made of postencephalitic symptoms. The diagnosis was left in doubt with the suggestion of paranoia or compulsion neurosis.

In the early part of 1936, she began to have trouble with her eyes, complaining that they would turn up in her head. The crises would occur about twice weekly, often lasting for an entire day.

In July, 1937, the patient met with a minor street car accident. Her condition was aggravated in that her fears became greater. She was seen again at the clinic, it being observed this time that her mental picture suggested a paranoid state. Mild physical findings of Parkinsonism were noted.

On April 9, 1939, the patient took two ounces of rat poison discovered to contain yellow phosphorus. She was admitted to the Temple University Hospital in a state of shock. She talked freely, saying she had decided to commit suicide, believing the family would be better off without her. She said that the neighbors were planning to kill her and her family. Suspicions that her neighbors were planning a murder for which she was to be blamed were common. She realized that these thoughts were "all foolish," that her neighbors were good to her, but she was unable to rid herself of these false ideas.

While at the hospital she experienced several oculogyric crises. At these times the patient had thoughts similar to those mentioned previously, but now was convinced of their actuality. Before a crisis, she felt someone was going to kill her husband, but realized that the idea was false. During a crisis, however, she became certain that someone was going to kill her husband. The mental picture seemingly changed from an obsessive syndrome to a paranoid state. Her mood was euphoric with periods of depression. She had recurring homicidal dreams, and frequently dreamed that her husband went to work without returning.

The essential neurologic findings were a fixed facies and some hyperactivity of the left side of the face, with impaired voluntary emotional responses. There was no defect of the extraocular move-

ments except for some loss of convergence. The pupils were dilated and reacted sluggishly to light. During an oculogyric crisis her eyes turned up and out, although she could bring them down momentarily. The crisis lasted from one-half to three hours. There was an abnormal muscle tonus as evidenced by cogwheel rigidity in the right arm, with impairment of fine movements in the left hand. All reflexes were essentially normal, as was the sensory status. With therapy which included a medical regimen for phosphorus poisoning plus psychotherapy, benzedrine and stramonium, there was definite improvement in all spheres. She was discharged May 11, 1939.

The mental picture in this patient was unusual. She was obsessed with the idea that others might harm her or hers. Ordinarily, obsessive patients fear that they may harm others. During the oculogyric crisis the picture was converted into a paranoid condition, as the patient became convinced of the reality of the delusional system, previously considered foolish. It seemed to the physicians that during a crisis the patient was able to express hate more freely and unequivocally. She would say, "I love the nurses, they are so good, but suddenly I feel afraid. I feel dissatisfied with them, and my eyes turn up."

The mental symptoms preceded all evidences of Parkinsonism, including the oculogyric crises. These, as far as could be determined, were the first signs of what seemed to be a postencephalitic state. Since the mental picture antedated all postencephalitic phenomena by at least two years, we may assume that the obsessive state was not related to the organic disease of the central nervous system. Schilder's[2] explanation that the obsessive syndrome might arise from a state of akinesia or muscle rigidity, therefore, is not pertinent here. However, it has already been noted that there seems to be a relation between the mental condition and the seemingly existing organic disease. Wexberg[3] points out that by a pathologic process a pattern of action and thinking of the compulsive type is touched off where the pattern normally should be free and responsive. In this patient the compulsive pattern was definitely established before there was any evidence of a postencephalitic state. Here, by a pathologic process a pattern of action and thinking of the paranoid type was touched off where it was orig-

inally obsessive. The progress of the illness, however, might be understood through a different approach. During childhood, the patient feared violence and rape. The mental picture occurred acutely after an actual attempt at sexual assault. Her first thought was of her disappointment in America. Fear of injury to herself led to a rage reaction, then obsessive phenomena exemplified in the belief that "the nurses are good, but they might hurt me." This was followed by the compulsive phenomenon in the oculogyric crisis and lastly by the delusion that the nurses were plotting against her, accompanied by additional hate. The final state was one of muscle rigidity. We recognize both the symbolic and the actual value this would afford as a protective mechanism, particularly against the release of aggressive trends. We, therefore, arrive at the possibility that the psychic tension resulted in the somatic changes manifest as Parkinsonism.

Case 2. B. V., a 27-year-old white married female, is said to have been a premature baby, her early development normal. She had influenza at the age of seven, during the 1918 epidemic. She was raised by an overly religious and critical aunt. The patient felt she could never satisfy the demands of this aunt. She has always been of a shut-in type. After a short courtship, she was married in 1936. Her married life was unhappy. One and one-half years later a child was born to her, to the displeasure of her husband. He thereupon insisted that she go to work to support the child. Within two months after delivery, she was employed as a waitress. One day while working she was overwhelmed by a feeling of extreme guilt, feeling she had done something wrong, and that people were staring at her. Suddenly her eyes turned upward and she was forced to leave work. This was the first of a series of oculogyric crises. During these spells, the patient would have the impression that people were making derogatory remarks about her, e. g., they criticized her for dressing nicely, or for taking her child to the movies even though she was receiving public assistance. At these times the patient had a feeling of nonexistence, of having "lost all sense of reality." She had some insight into her condition, saying that the voices must be imaginary. She first came under treatment in June, 1939, with the chief complaint of weak-

ness and slowed muscular activity. She stated that these had been troubling her for only the past month.

The outstanding physical findings were slow gait, some impairment of associated movements of the arms, and sluggish pupils with difficulty in ocular movements particularly in upward gaze. Her speech was slow and monotonous.

With stramonium and psychotherapy, the patient showed much improvement. She has adjusted fairly well to an existence apart from her husband and is supporting her child. There is no evidence of abnormal mental trends except at the time of an oculogyric crisis.

During treatment, the patient expressed obvious hostility to her husband. When the child was born and she was forced to go to work, the hostility toward her husband was extended to the child in an ambivalent manner. In one of her earliest crises, she felt that people were accusing her of desiring to get rid of the baby. The crisis was preceded by ideas that someone was criticizing her. These thoughts would occupy her attention even though she tried to dismiss them as having no basis in fact. During the crisis, however, these thoughts of criticism became true ideas of reference, leading to more hostility, finally to muscular rigidity.

Discussion

Two cases have been discussed showing minor physical findings usually referred to as representing a postencephalitic state. In both these patients, the oculogyric attacks were the first evidence of Parkinsonism. This is unusual, and Jelliffe[1] states, "Most observers record that these crises have been observed only in those postencephalitic patients who have developed more or less completely the Parkinsonian syndrome." In the 200 or more cases abstracted by Jelliffe, the onset of the oculogyric movements took place somewhere between the general limits of four weeks to seven years after the appearance of postencephalitic symptoms. In case 1 no complaint was noted referable to the extrapyramidal rigidity, while in case 2 the complaint was of only short duration compared

to the duration of the crises. The physical findings of Parkinsonism in both cases were slight and had been overlooked by other physicians. In neither case was a history of any type of encephalitis obtained.

In patient 1 an actual attempt at rape renewed childhood fears resulting in obsessive symptoms followed by what seemed to be a postencephalitic state. In patient 2, childhood fears of an aunt were renewed by a domineering husband. Under the added stress of the birth of an unwanted child, the patient expressed her rebellion in the form of obsessive symptoms followed by physical findings usually referred to as Parkinsonism. Jelliffe particularly has stressed the fact that oculogyric crises cannot be dismembered from other synergistic movements such as those of position, speech and even those which are "purely psychic," except for theoretical purposes. The writers are in perfect accord with his interpretation, "In short, the song of the psalmist, 'I will lift up mine eyes unto the hills, from whence cometh my help,' is the sense of an ethical compulsory substitute for the emergence into consciousness of repressed tabooed wishes, as the primitive positive level in ethical structure which has built up civilization and culture and which is believed to have some bearing on this study of compulsive activities." The relation of the emotional factors to the neural pathology in these cases is not clear. They are best understood, however, as emotional problems. The sequence of events in the illness observed was as follows: poorly managed hostility in the patient, obsessive ideas, followed by compulsive phenomena in the oculogyric crises with further hostility, paranoid ideas, and finally muscle rigidities.

Summary and Conclusion

Two cases presenting compulsive phenomena associated with oculogyric crises have been presented since it was believed that the type and sequence of the symptomatology might offer insight into the psychopathology.

The transition of the mental picture from an obsessive to a paranoid state occurring during an oculogyric crisis was considered sufficiently unusual to merit description.

The postencephalitic states, especially where physical findings are slight and there is no history of infection, cannot be understood solely as a psychiatric problem or solely as a neurologic disease. They are psychosomatic problems open for further study and investigation.

Psychiatric Department
Temple University
Philadelphia, Pa.

REFERENCES

1. Jelliffe, Smith Ely: Psychopathology of Forced Movements in Oculogyric Crises. Nervous and Mental Diseases Monographs, New York.
2. Schilder, Paul: The organic background of obsessions and compulsions. Am. Jour. Psychiat., 94:6, May, 1938.
3. Wexberg, Erwin: Remarks on the psychopathology of oculogyric crises in epidemic encephalitis. Jour. Nerv. and Ment. Dis., 85:56, 1937.

INSULIN SHOCK THERAPY IN THE OLDER PATIENT WITH SCHIZOPHRENIA OR A SCHIZOPHRENIC-LIKE REACTION

BY G. WILSE ROBINSON, JR., M. D.

The several indications for the various forms of shock therapy have not yet been standardized. A trend seems to be growing which shows that insulin shock (Sakel technique) is of value primarily in the schizophrenic and schizophrenic-like reactions, while metrazol shock and related procedures are indicated in the affective reactions, especially the chronic affective psychoses of the climacteric and presenile years.

This concept is based upon recent reports and statistical analyses. Ross' latest report,[1] a two-year followup, notes a 50 per cent improvement in a series of 1,039 cases treated with insulin shock. This series includes all types of cases of all ages and all periods of duration. Of the total series 37.3 per cent are still in the community, and 58.4 per cent are in mental hospitals. The rest are unknown or dead. The "duration of condition" breakdown shows that 61.6 per cent of those cases treated during the first six months of illness are in the community, while 47.7 per cent of those treated during the second six months of illness are well enough to be home. Heilbrunn and Sternlieb[2] report that 315 schizophrenic patients treated during a period of almost three years showed 72 per cent recoveries and 10 per cent social remissions in cases with a duration of psychosis of less than seven months; 35 per cent and 20 per cent respectively for those ill from seven to 18 months; and 10 per cent for cases of over 18 months duration. A similar percentage was obtained in patients who had not responded to previous metrazol therapy. There had been so far (March, 1940) only 15 per cent relapse in the first two groups. These writers also reported that the treatment with insulin and metrazol simultaneously did not benefit patients who had failed to respond previously to either therapy alone. They discontinued this modification for several reasons.

Ross and Malzberg[3] concluded that metrazol produced even fewer recovered cases (1.6 per cent recovered and 9.9 per cent much im-

proved of a total metrazol-treated series of 1,140 patients) than were found in cases not treated by any shock therapy. (In a control series there were 3.4 per cent recovered, 11.2 per cent much improved.) This viewpoint is confirmed by a cooperative report on 659 patients, published in Germany,[4] analyzing 560 cases treated with insulin, 132 with metrazol, and 67 with both. The insulin series showed 37.7 per cent symptom-free and 28.9 per cent improved. The metrazol series showed 18.9 per cent symptom-free and 37.1 per cent improved. W. Menninger[5] reports that only 17 per cent of his series of schizophrenics showed a social remission after a course of metrazol therapy.

Results at the Neurological Hospital, Kansas City, Mo., in a series of 12 schizophrenic patients treated with metrazol, showed: Three made complete recoveries, two were sufficiently improved to go home, three improved slightly, and four did not improve. However, only two of the improved patients have maintained their improvement, so that 85 per cent of this series are in a state hospital or at home, completely incapacitated. Since these patients were treated some months ago during the period when metrazol was being tried extensively, enough time has elapsed to show that in this small series metrazol was not an effective treatment in schizophrenia if sustained results are desired. In the affective reactions, especially in the depressive phases and types, metrazol convulsive shock at times seems to be almost a specific. Bennett,[6] Young and Young,[7] Cottington and Gavigan,[8] and Hackfield and Halvorsen[9] have all reported highly favorable and sustained results.

Wilson[10] reports that 78 per cent of his patients diagnosed involutional melancholia have been able to make a social and business adjustment after a course of metrazol convulsive shocks. The writer's series of involutional and presenile depressions and melancholia show, after a six months followup, that 75 per cent have made and continue to make an excellent social adjustment. When we keep in mind that Palmer and Sherman[11] found that only 30 per cent of their patients diagnosed involutional melancholia were able to make a satisfactory adjustment after many months of hospitalization, metrazol therapy seems indicated in this type of abnormal reaction in spite of the potential risks.

In recent months at this hospital, two cases admitted at about the same time have raised a problem which does not seem to have been discussed in reports on shock therapy.

Case Reports

Case 1. D. S., age 48, was admitted to the Neurological Hospital in a state of profound melancholia and depression. About 18 months before admission, her stepfather had died without a will and had left a considerable estate. This estate was in litigation throughout the period before admission, and was a constant source of worry and irritation to the patient. Although she ate well during the first year following her stepfather's death, she lost weight gradually. Three months before admission she nearly ceased eating, and her weight at the time of admission was 101½ pounds, a loss of about 60 pounds from her normal weight. Physical examination revealed profound emaciation and dehydration, and the skin presented evidence of clinical pellagra. Psychiatric examination revealed an uncooperative, quiet, almost mute individual whose only conversation related to death and destruction of herself and family. She cried easily without apparent provocation. Most of the time she lay staring into space, preoccupied with her thoughts. Laboratory examination revealed a negative Wassermann and a glucose tolerance reading of 108.9 fasting; 203.7 one-half hour; 223.7 one hour; 234.3 two hours; 299.6 three hours; and 299.4 four hours.

A tentative diagnosis was made of profound malnutrition with clinical pellagra superimposed upon a psychosis of undetermined type, probably involutional melancholia. Vitamin therapy, forced feeding, and 10 per cent glucose infusions with insulin, 1 unit to each 5 gm. of glucose, were undertaken at once. In two weeks the patient's malnutrition was somewhat relieved. She had gained 12 pounds and the skin lesions were clearing up. However, the depression and melancholia were still present in as marked a degree as ever, and the patient denied any subjective feeling of improvement. She was still preoccupied with thoughts of death, and at times appeared confused and disoriented. She did not seem to have any delusional-hallucinatory-like symptoms of a fixed or consistent type. She did demonstrate ideas of reference occasionally.

The diagnosis of involutional melancholia was felt to have been confirmed, and metrazol therapy was started. During the next four weeks she had nine full convulsive shocks. After the first three or four, she became more cooperative and accessible. She said that in some ways she felt better, while in other ways she did not. She would not enlarge upon this statement. Her preoccupations and ideas of reference continued, and she still took no interest in her appearance or social contacts. During the next two weeks she showed no improvement. Certain paranoid delusions began to manifest themselves, and she complained of having had "dreams" both while awake and during sleep. As it has been the writer's experience that the involutional melancholia patient should improve steadily under metrazol, it began to seem that the original diagnosis made in this case might not have been accurate. As time passed, the schizophrenic elements became increasingly manifest. Six weeks after the patient's admission and four weeks after the initiation of metrazol therapy, the decision was reached to discontinue metrazol and to undertake insulin shock treatment. As soon as insulin therapy was started, the patient began to improve. She had 15 full insulin shocks in all, and was discharged two and one-half months after admission in a state of social remission. She was still somewhat fearful of the future, but demonstrated no delusions, ideas of reference, preoccupation, restlessness, mutism or confusion. It would have seemed desirable to continue treatment, since it was felt that a full remission might have been obtained, but financial reasons forced the discharge. A glucose tolerance test taken before insulin shock was started showed fasting 102.6; one-half hour 174.7; one hour 125; and two hours 131.5—indicating that the malnutrition element had been eliminated without recovery of the patient, hence that this was a secondary rather than a primary etiological factor.

Case 2. M. M., age 50, was first seen at home two weeks after the accidental death of her favorite son-in-law. At that time she was profoundly depressed, mute; and insistently refused food and water. She showed marked evidence of dehydration and acidosis. Her sister-in-law was a graduate nurse who had had psychiatric nursing experience. Because of this and the limited finances, it was decided to leave her in the home environment temporarily

while nutritional and hygienic measures were instituted. She was given tube feedings, and 10 per cent glucose infusions with insulin and vitamins. After one week of this program she became worse, her physical condition had further deteriorated, and she was admitted to the hospital. She manifested a complete suppression and depression of all voluntary physical activity. There was a complete lack of cooperation. The Wassermann test was negative. A glucose tolerance test showed fasting 108.3; one-half hour 192.8; one hour 266.7; two hours 345.2; three hours 223.4; and four hours 172.6. Because the nutritional problem was acute and almost insurmountable in her condition at that time, she was given three metrazol shocks in rapid succession (at two-day intervals). After the second she became more cooperative, ate when fed, and took sufficient fluids. She responded to questions and occasionally volunteered conversation. However, she was still resistive at all times. Because of the improvement, metrazol was continued. She began to show a slight interest in her surroundings, and after eight full convulsive shocks became more cooperative, moderately active and talkative, but was still fearful and seclusive most of the time. Only occasionally would she volunteer conversation. After the ninth convulsion she relapsed into a constant state of seclusion, demonstrating fear of everyone and everything. At about this time she told one of the members of the staff of some "bad dreams" which she had had for several weeks. She was constantly seeing fearful monsters which were going to eat her. She would never discuss them again, but her actions demonstrated that she was still experiencing these hallucinations. Continuation of metrazol therapy produced no mental improvement. Nor did her physical condition improve, and after the eleventh shock it was concluded that the treatment procedure should be changed. Insulin shock was started, and she improved slowly during the first week of this treatment. After the fourth full insulin shock, she began to improve rapidly. She was discharged eight weeks after the initiation of insulin therapy, having had 51 reactions. Apparently she was in a state of full remission. Metrazol shocks were given at infrequent intervals during the first three weeks of insulin therapy, then were discontinued. Her glucose tolerance curve did not return to normal, but was markedly improved.

The favorable response of these two patients to insulin shock therapy, after other measures, including metrazol, had failed, led the writer and colleagues to a recheck of material. Eleven patients over 35 years of age were found who had reactions with a heavy preponderance of schizoid-like symptoms, and who had been treated with insulin shock therapy. Most of these patients had been treated before Bennett had made his original report, hence before metrazol was used freely in the older patient. One of these patients had followed the same course as the two cited above in that she had been given a full course of metrazol convulsive shock before her admission, with virtually no clinical improvement, yet went forward into a full clinical remission under insulin shock. One other patient was given metrazol after insulin shock had failed, but without clinical effect whatsoever.

Patient	Year discharged	Age	Duration in months	Months in hospital	Number of shocks	Type of treatment	Present condition*
M. B.	1937	36	60	2½	56 I	Insulin	F. R.
H. S.	1938	42	4	2—	28 I	Insulin, then	N. C.
					8 M	Metrazol	
F. O.	1938	39	1	2—	40 I	Insulin	F. R.
V. O.	1938	45	8	2	52 I	Insulin	F. R.
H. M.	1938	49	1	1½	24 I	Insulin	F. R.
I. W.	1938	42	12	1+	29 I	Insulin	S. R.
M. M.	1938	36	1	1½	28 I	Insulin	F. R.
P. C.	1938	41	18	2	38 I	Insulin	F. R.
J. B.	1938	37	3	1½	30 I	Insulin	F. R.
W. H.	1939	36	4	2—	28 I	Metrazol, then	F. R.
						Insulin	
G. N.	1940	39	1	2	40 I	Insulin	F. R.
D. S.	1940	49	3	2½	9 M	Metrazol, then	S. R.
					15 I	Insulin	
M. M.	1940	50	1	3	18 M	Metrazol, then	F. R.
					38 I	Insulin	
Averages		41.6	9	2	35.5 I		92.3

*Footnote: N. C.—No change.
S. R.—Social remission.
F. R.—Full remission.

SUMMARY

The excellent results in this series, 92.31 per cent full or social remissions as the immediate result, and 90 per cent full sustained social remission after one and one-half years, are significant in indicating that insulin shock does have a place in the total therapy of the older patient with schizoid-like reactions, even though the predominant picture be that of the affective type.

The older schizophrenic has not been analyzed as a group in any reports. The writer and colleagues feel that, with the breakdown of the two types of shock therapy into distinct indications, which seems to be developing, this group should be given every diagnostic consideration. Metrazol therapy is not the sole treatment in this group, any more than it is the treatment of choice in the younger age group. Metrazol convulsive shock is an effective method for returning the simple affective disorders to normal, but when the presented psychosis of the middle-aged patient is schizophrenic, schizophrenic-like, or presents schizophrenic residuals after the affective abnormalities have been eliminated by metrazol, then insulin shock is an efficient therapeutic agent.

An exact prognosis for shock therapy as applied to the individual patient before treatment is started has been difficult. Exact criteria have not as yet been worked out and standardized. From these results it would seem that the older patients with schizophrenic elements in their psychosis have a much better prognosis than the younger patients. These patients had all made a partial or apparently complete adjustment to their environments during their adult years. The schizophrenic process apparently was latent, and manifested itself for a variety of reasons in these patients at this time. But the very fact that it was latent during these many years indicated that it might be suppressed easily once more, and results at this hospital confirm this viewpoint. Rupp and Fletcher[12] found that the age of onset of the schizophrenic had little bearing on the outcome from the standpoint of "spontaneous" remissions, except that those having an onset at the age of 45 or later tended to do less well than those having an onset at a younger age. This concept is contrary to the writer's findings. While most of his patients were under 45, those who were over this age at the time of onset seemed to do as well as the others in this series.

Conclusions

1. This series of 13 patients who, at the age of onset, were in the upper age limits for onset of schizophrenia, gave 92.31 per cent excellent results for immediate recovery rate, and 90 per cent excellent results for the one and one-half year followup, following insulin shock therapy.

2. The older patient with schizophrenic elements in his psychosis should be treated with insulin shock therapy.

3. Prognostic criteria for the individual patient when insulin shock therapy is proposed should take into consideration the type and quality of the adult adjustment before onset, the age of the patient at the onset of the symptoms, in addition to the other factors which have been proposed, such as duration of illness before treatment and the type of symptoms presented.

Neurological Hospital
Kansas City, Mo.

REFERENCES

1. Ross, John R., et al.: A statistical study of results of pharmacological shock therapy in the New York State hospitals. Read before the American Psychiatric Association, Cincinnati, O., May 23, 1940.
2. Heilbrunn, Gert, and Sternlieb, Ruth: Insulin therapy of schizophrenia in the Elgin State Hospital. Am. Jour. Psychiat., 96:1203, March, 1940.
3. Ross, John R., and Malzberg, B.: A review of the results of the pharmacological shock therapy and the metrazol convulsive therapy in New York State. Am. Jour. Psychiat., 96:297, September, 1939.
4. Results of shock therapy in schizophrenia. Berlin correspondent. J. A. M. A., 112:2619, June 24, 1939.
5. Menninger, W. C.: An evaluation of metrazol treatment. Bull. Menninger Clinic, 4.95, July, 1940.
6. Bennett, A. E.: Convulsive shock therapy in depressive psychoses. Am. Jour. Med. Sci., 196:420, September, 1938.
7. Young, R. N., and Young, G. A.: Treatment of the psychoses with induced hypoglycemia and convulsions. J. A. M. A., 112:496, February 11, 1939.
8. Cottington, F., and Gavigan, A. J.: Metrazol treatment of depressions. N. E. Jour. Med., 220:990, June 15, 1939.
9. Hackfield, A. W., and Halvorsen, C.: Modified convulsive therapy for involutional depressive psychoses. Northwest Med., 38:373, October, 1939.
10. Wilson, David C.: The results of shock therapy in the treatment of affective disorders. Am. Jour. Psychiat., 96:673, November, 1939.
11. Palmer, Harold D., and Sherman, Stephen H.: The involutional melancholia process. Arch. Neurol. and Psychiat., 40:762, October, 1938.
12. Rupp, Charles, and Fletcher, E. K.: A five to 10 year follow-up study of 641 schizophrenic cases. Am. Jour. Psychiat., 96:877, January, 1940.

BOOK REVIEWS

Psychotherapy. By LEWELLYS F. BARKER, M. D. 218 pp. New York and London. D. Appleton-Century Company. 1940. Price $2.00.

Dr. Barker is well known as one of a relatively small group of internists who have gained a deep insight into psychiatric problems and who participate with psychiatrists in their point of view. Long a distinguished teacher of medicine and a student of the mental life, he is particularly well qualified to discuss the subject of psychotherapy, from his own experience and from his familiarity with general medicine and psychiatric literature.

The first impression, upon reading this book, is one of the author's breadth of view and of his painstaking care in the preliminary study and planning of a course of treatment best suited to the patient's individual needs. His is a thoroughly catholic approach to treatment; psychotherapy is one form which he applies when indicated. He maintains an even balance, not expecting the impossible of mental therapy, nor hesitating to employ it when indicated. He defines psychotherapy as "treatment that attempts to improve the condition of a human being by means of influences that are brought to bear upon his mind."

It will be interesting to learn how Professor Barker deals with psychoanalysis and the work of Sigmund Freud. This he does in a section of 16 pages, stated fairly and without prejudice, giving first a brief history of its origin and later on setting forth an outline of the Freudian psychology. It is evident, however, that he does not accept considerations of the Oedipus complex and of infantile sexuality at the full value accorded them by the Freudians. He gives greater approval to the position of Adolph Meyer and to psychobiology. Of these he speaks with definite approval, and he defends Meyer against some of the criticisms of Schilder. It is noteworthy, however, that he gives more space to the discussion of Freud's views than to all other systems of psychotherapy added together. This is in accordance with the observations of others doing work in this field. Though they may differ with Freud on some points, the fundamental Freudian psychology, modified though it may be by other practitioners, is the foundation of modern treatment by influencing the mind.

This book heartily merits approval. It should be in the hands of every practising psychiatrist, and medical men generally may read it with understanding and profit.

The author's position is succinctly stated in the closing sentence, which is worthy of repetition for its truth and practicability: "Meanwhile, it is our duty to apply to our patients all forms of treatment which we think will be helpful, and, in the treatment of the neuroses and psychoses especially, psychotherapy is likely for a long time to continue to occupy a dominant place."

Psychiatric Dictionary. With Encyclopedic Treatment of Modern Terms. By LELAND E. HINSIE, M. D., and JACOB SHATZKY, Ph.D., New York. The Oxford University Press, 1940. Cloth. 559 pp. Price $10.50.

We have waited long for an accurate and comprehensive psychiatric dictionary, one in keeping with the present-day importance of psychiatry as a member of the group of biological sciences. The preparation of such a reference book is a major undertaking, requiring as it does almost endless reading, notation and deliberation. Such a book, the product of long days and evenings of work, is now fresh from the press; it is near to the ideal and reflects credit upon the scholarly attainments of the authors and their corps of collaborators.

Dr. Hinsie has assumed responsibility for the definitions of terms in use in psychiatry and related sciences, and Dr. Shatzky for the philological part of the dictionary. Both are admirably qualified for their parts in this undertaking; the former is professor of psychiatry in the College of Physicians and Surgeons of Columbia University, and the latter is research librarian in the New York State Psychiatric Institute and Hospital.

The pages are printed in two columns, which is more economical of space; the terms are printed in bold face type, followed by the phonetic pronunciation, the part of speech, as noun or adjective, the philological derivation, and finally, the definition. Pains have been taken to make the latter comprehensive and understandable; the word "cathexis," for example, is defined in 52 lines and "symbolism" in nearly 100 lines. Other terms are defined more briefly; a few by just a line or two or a few words.

One commendable feature of this dictionary is the ease with which the reader may refresh his memory regarding a little-used term which may have escaped his mind. For example, "Taphophilia" is defined as "The morbid attraction for cemetaries." The term may readily be found by reference to its popular English equivalent "cemetary, attraction for a—Taphopilia." Many other phobias and manias are similarly arranged.

To a large extent the authors have adopted the plan of quotations from standard authors who have defined the term or used it in such a way that the meaning is clearly evident. Credit is given in each case to the author

and to the publisher of the book. While this plan is desirable for some purposes it makes the definitions sometimes unnecessarily long. As far as the opinion of this reviewer is concerned, he would prefer to receive Dr. Hinsie's studied and well formulated definition than a quotation from the writings of D. Hack Tuke whose well known dictionary of psychological medicine was popular 50 years ago. This brings up another question of capital importance in the construction of a psychiatric dictionary.

The authors in their preface inform us that, "The dictionary comprises all important terms and concepts used during the span of time approximately since Hippocrates up to our own day." They plan to mark as obsolete those terms which are losing currency or are no longer in use, but that plan is not adhered to in every instance. A considerable number of terms, not all of them going back to the days of Hippocrates, but going back to the nineteenth century, might be eliminated from modern psychiatric literature with benefit. Some of them no longer mean anything from our present conception of the nature of mind and mental processes, others have been definitely superseded by better terms and should be abandoned. Examples are the numerous forms of delirium mentioned when the definition indicates quite clearly that delusion is meant. DELIRIUM GRANDIOSUM defined as "megalomania," DELIRIUM PERSECUTIONIS, which is defined as "paranoid condition." Neither of these is indicated as obsolete. In all there are about three pages of deliria, many of which are really now only psychiatric curiosities.

It is to be presumed that the reason for retaining so many obsolete and archaic terms was to make the dictionary of value to research scholars who delve into the musty tomes of the middle ages in historical research. While that may be an admirable motive, the result is likely to prove confusing to a present-day psychiatric student and is likely to lead to controversy between editors and contributors of psychiatric papers over the continued use of such terms as "general paralysis" and "insanity," which appear to carry the sanction of this latest and most complete of psychiatric lexicons. As to these two terms, efforts are being made to eliminate the former as a misnomer and to substitute for it the more accurate if somewhat colloquial term "general paresis" or better still the scientific term "meningoencephalitis." It is true that "general paralysis of the insane" is not defined and that reference is made to general paresis, but the obsolete character of the former is only to be inferred, and the scientific term does not appear. As for the term "insanity," it is admirably defined with emphasis upon its medico-legal implications, but it appears in other references without restrictions as to its use. INSANITY, ACUTE, CONFUSIONAL, is defined as "Primary confusional insanity;" INSANITY, ALCOHOLIC, is defined as "In-

sanity due to alcoholism;" INSANITY, CLIMACTERIC, is defined as "Insanity due to menopause;" INSANIA LACTANTIUM is defined as "lactational insanity," and there are many others in the three or four pages devoted to definitions of this term. They are doubtless examples of *lapsus calami* to be corrected in the next edition.

The book as a whole is so admirably done that it may seem somewhat captious to refer to these instances; but the duty of the reviewer to be impartial and the demands for accuracy in psychiatric speech and writing make such comments necessary.

The Oxford University Press is to be congratulated upon its share in making this useful reference work available to libraries and private readers; the book deserves, and it undoubtedly will have, an extensive circulation.

Psychological and Neurological Definitions and the Unconscious. By SAMUEL KAHN, M. D., Ph.D. 148 pages, with extensive bibliography and index. With an introduction by M. Mortimer Sherman, M. D., Boston. Meador Publishing Company, 1940. Price $2.00.

The book has been prepared, so the author informs us, to help bridge the gap between the work of the neurologist and the psychiatrist, that between the work of the psychiatrist and the psychologist, and that between the interests of the psychiatrist and the psychoanalyst. Also he sees too wide a gap, which he would bridge, between the psychiatrist and the endocrinologist, one between the physician and the mental healer, another between the armchair philosopher and the clinical psychologist, and finally one between the laboratory worker and the artistic healer.

It is no small task that the doctor sees before him and he is not so sanguine as to believe that it can be accomplished in one small volume. His contribution to such a worthwhile undertaking seems to be the defining of terms which are in common use among all of these groups. He also contributes an essay, "The Philosophy of the Unconscious and Psychoanalysis," and devotes a chapter to the history and background of Sigmund Freud and the psychoanalytic movement. Before proceeding to his glossary of terms he takes advantage of the opportunity to insert a chapter entitled "Subconscious Speculations."

The glossary, which occupies half the volume, includes terms from neurology, psychiatry and psychoanalysis. Those which pertain to psychoanalysis are, in the main, very good. Dr. Kahn has gained some distinction in this field and has made some worthwhile contributions. Some other definitions, however, leave something to be desired. For example, dipsomania is defined as "The compulsion to drink alcoholic concoctions." This

definition, of course, is inadequate; it does not sufficiently distinguish dipsomania from other alcoholic habituations. Another short definition which fails to make the term clear to the uninitiated reader is "dystonia" which is defined as "A pathological condition of muscular tonicity." This fails to point out the direction in which the muscular tonicity is disturbed. He defines echolalia as "A state of mind where the patient repeats, etc." Echolalia is a noun; it is a phenomenon, the act of repeating words and phrases. The state of mind of which this symptom is an expression is catatonia.

Dr. Kahn does not conceal his admiration for Sigmund Freud; the book is dedicated to him; and references throughout the text are laudatory. The chapter which he devotes to the history and background of the psychoanalytic movement is interesting and contains material from various sources which it is well to have preserved in available form.

This little book might well have a place in every psychoanalytic library.

The Hypothalamus and Central Levels of Autonomic Function. By The Association for Research in Nervous and Mental Disease. Williams and Wilkins Co., Baltimore, 1940. 1010 pages. Price $10.00.

This volume is the twentieth research publication of the Association for Research in Nervous and Mental Diseases. It is the result of the combined efforts of 42 contributors who are outstanding in their fields and is edited by J. F. Fulton, S. W. Ramon and A. M. Frantz. A year before the meeting a précis on preoptic, hypothalamic and hypophysial terminology was circulated to the contributors. On their part, the writers have made a serious effort to conform to the terminology suggested; and this has overcome much of the confusion in the description of their subject.

The main text is preceded by a historical introduction, including Frohlich's original text describing the adiposogenital syndrome.

The text is divided into three principal parts: The anatomy of the hypothalamus, which includes the embryology and comparative anatomy; the physiology of the hypothalamus; and a clinical symposium with case histories.

Following each chapter, is a discussion and a brief bibliography. At the end of the volume, are references to the literature from various papers, assembled in a single alphabetical list comprising 68 pages. This probably represents the most complete bibliography of diencephalic literature thus far compiled.

There are numerous photomicrographs, photographs of gross specimens, drawings and diagrams of excellent quality.

This volume undoubtedly represents the most comprehensive, detailed collection of experimental and clinical work on the subject thus far published; and it is highly recommended by the reviewer.

Psychiatry for the Curious. By GEORGE H. PRESTON, M. D. Illustrated with sketches by the author. New York and Toronto. Farrar and Rinehart, Inc. 1940. Cloth. pp. 148. Price $2.50.

Here is a book that is better than its title would lead one to expect. Written for lay readers, there are almost no technical words not made clear as to meaning and use, and the style is simple and flowing. So many books intended for professional readers are being written on psychiatry under the guise of mental hygiene by non-psychiatrists and often by non-medically trained writers that it comes as a pleasant surprise to find a book on psychiatry intended for laymen written in their language and for their understanding by a distinguished psychiatrist and hospital administrator. Still more surprising is it, to find that a book of this sort can be made free from sensationalism and made sound in theory and example.

Briefly stated it is a presentation of the principles upon which modern psychiatry is based; environment as it influences the child and man, the formation of behavior patterns, how such patterns influence the attitude toward people and life generally. Psychoneurotics are described as people who suffer from imaginary pains or hurts, as real to them as real pains or hurts would be. These pains and hurts serve to shield the individual against becoming conscious of something still more painful but of another sort, and so they are in a measure a defense. Psychoneurotics must succeed in fooling themselves if their defenses are to work. They hang on to their symptoms in the same way that a man with a broken leg hangs on to his crutch. "If we treat the crutch and not the leg he will never get well."

The paranoic group is introduced by reference to the man who stumbles over the cat and instead of kicking himself for being inattentive and stupid, kicks the cat. "Defense by Cat Kicking" is a type of reaction which Dr. Preston develops into projection mechanisms, hallucinations and delusions of grandeur and persecution. In the same breezy style he takes up the principal reaction types as the schizophrenics and cyclothymics and others; but, employing quaint terms of his own; he says: "Somewhat the same terms I might use to present a baseball game or an account of the most recent filling station holdup."

There will be many people grateful to Mrs. Preston "because without her constant help it (this book) would have remained two baskets full of scrap paper."

Practical Clinical Psychiatry—5th Edition. E. A. STRECKER, M. D. and F. G. EBAUGH, M. D. The Blakiston Co., Philadelphia, 1940. 678 pp. with glossary and index. Price $5.00.

This deservedly popular textbook continues to progress, with this, the fifth edition, appearing two years after the fourth. The fundamental material, including the chapter "Psychopathological Problems of Children" by Dr. Leo Kanner, has been retained. The division of the material into reaction types for descriptive purposes in the main text is continued. The major changes from the fourth edition are three. In the chapter entitled "Psychobiological Conceptions," a summary of the work of Vigotsky, Kosanin, Piaget, Cameron and others on the thinking process and its connection with psychiatric production has been included. A short discussion of "Crowd-Minded Behavior and Mass Reactions" has also been added to this chapter, being adapted from the 1939 Salmon Lectures, "Beyond the Clinical Frontiers." This short exposition of the subject and the plea for social-mindedness in psychiatry is particularly timely.

Under "the organic reaction type," the section on neurosyphilis has been re-written to bring it more in line with present-day clinical-pathological conceptions. The section on alcohol is also expanded somewhat to include a brief dissertation on symptomatic alcoholism. Other recent advances discussed are those of electroencephalography, particularly in relation to epilepsy, vitamins, as related to the psychoses occurring with pellagra, and the drug therapies in the functional psychosis—amytal, metrazol and insulin—as well as leucotomy in the manic-depressive psychosis. The bibliographies of several chapters have been expanded. Aside from these additions in the material discussed under the various headings, there are no major changes.

So often are textbooks criticized for presenting material already outmoded by recent developments that it is a distinct pleasure to find this one remaining abreast of the field. This edition, as with the previous one, can be recommended to students of medicine and psychiatric social work, as well as to the general practitioner, as a foundation for knowledge in psychiatry.

Sex in Development. By CARNEY LANDIS and CO-AUTHORS. Paul B. Hoeber, Inc., New York and London, 1940. 234 pp. with appendices, bibliography, author index and subject index. Price $3.75.

As its sub-title indicates, this volume reports "a study of the growth and development of the emotional and sexual aspects of personality, together with physiological, anatomical and medical information on a group of 153 normal women and 142 female psychiatric patients." The presentation

follows the custom of scientific tracts. There is a short introduction, giving a resume of current knowledge and theory on the subject, followed by a description of the materials and methods of the investigation. The results are then discussed and related to various sub-headings of the original topic. The work concludes with a brief, concise summary. The statistical material, with its mathematical evaluation, is given in tabular form in the various appendices.

The authors have attempted to maintain an objective factual attitude, with a minimum of inference and theorizing. In this they have succeeded while they are presenting their material; but the three final chapters (exclusive of the summary) are concerned almost entirely with inferences drawn from the authors' material, discussed in relation to various theoretical conceptions common in psychiatric circles today. In these chapters, the reader's attitude toward the text will probably be determined by the particular school of thought to which the reader is partial. Aside from these chapters the work is definitely factual and in its entirety is highly interesting. It is a definite contribution to that ill-explored field of feminine sexuality, its development and expressions.

The method of the investigation consisted of the personal interview on the conscious level, the questions for which appear in Appendix I. As the authors suggest, this method will attract criticism. However, it was used, as the only practical one to yield sufficient data on many individuals in a reasonable time. Each woman interviewed was also examined carefully by expert gynecologists whose findings were recorded on specially prepared forms, a sample of which also appears in Appendix I. Roentgenograms of the pelvis of each woman were studied and the pelvic types classified. Health records were also obtained. Thus it is that the types of data studied include psychological, anatomical, physiological and medical. All this material was then correlated and "quantified" according to methods outlined by the authors. The results are placed in tabular form in the various appendices under the general headings of vital statistics, evaluation scales and interscale relationships. The body of the book is concerned with a presentation of this material in expositive form under such sub-divisions as, among others, early background, adolescence, adult sex practices, physiological and anatomical factors, the psychiatric patient, the homoerotic woman, and the psychopathic personality. In short, the book represents a well-rounded scientific study, with the methods and material completely presented.

Starting in an attempt "to evaluate the importance of psychosexuality in psychopathology," the authors found that "the original problem divided and sub-divided itself until it was no longer recognizable, and the data

could not be arranged to afford a clear answer to the original problem that had been formulated." Though this be true, it in no way negates the value of the research, for it has produced much to verify previously expressed theories, much to question the validity of some concepts, and much to indicate problems for further research. A few examples chosen at random will illustrate these conclusions. Early home background was found to be of considerable importance in adult adjustment. More abnormal women than normal—and more poorly adjusted than well adjusted individuals among the normal—reported unfavorable early environmental factors. Masturbation and crushes between adolescent girls were found to be part of the normal course of development. Sexual adjustment in marriage was found to be the resultant of many components and not to be related to any single factor or group of factors. Anatomical and physiological factors in sexual function seemed less significant than psychological factors.

The psychosexual development of psychotic patients was found to deviate but slightly from the pattern of normal psychosexual development. Neurotic women showed significant variations in psychosexual development from the psychotic and the normal. Little difference was found between the psychosexual development of manic depressive patients and that of dementia præcox patients. Personality patterns were established, which, in the opinion of the authors, should be of more aid in therapy than the customary diagnostic categories. The sex education of youth was found to be almost universally unsatisfactory, though not prejudicial to adult adjustment. These are but a few of the points made and documented in the volume. In material such as this lies the chief value of the book. In the words of N. D. C. Lewis, who contributes a foreword, "such material is of value, not only for itself but for the information which it affords for additional research. It (this book) represents the type of systematic study desirable and necessary to bring order into this controversial . . . field of medicine and science."

Psychology and Psychotherapy. By William Brown, D. M., D. Sc., F. R. C. P. The Williams and Wilkins Company, Baltimore, 1940. 260 pages. Price $4.75.

At the present time it can be safely stated that psychoanalysis ushered in a new era in psychology and psychotherapy. Despite the still present resistance of organicists (who seem to be largely lacking in insight), the basic truth of the theory of the unconscious and of psychogenesis has invaded progressive psychiatric thought. This is not to say that psychoanalysis is now uncritically accepted. On the contrary, the contributions of psychoanalysis are admitted most grudgingly; unconscious resistances

continually crop up in the form of modifications, half-acceptance and attempts at compromise. In England particularly, leading psychiatrists, whose immanent objectivity coupled with unconscious resistance prevented either a complete denial or total acceptance of psychoanalysis, have become the chief exponents of an eclectic school which appears to practice a modified form of analysis yet resists a complete acceptance of the theory. Of this group, Dr. Brown is not the least worthy member.

"Psychology and Psychotherapy" is thus the personal expression of the author's ideas and technique, representing as it were his own attitude toward psychoanalysis not only in psychotherapy but in the larger framework of psychology. However, the book is more than merely a presentation of modified psychoanalysis. It is also an attempt to integrate psychoanalysis with older psychological contributions. Thus the early chapters are devoted to a short exposition of the theories of psychology on which Brown's credo is based. His theory of the neuroses rests first and foremost on the foundation of "dissociation." Although the theory of dissociation is largely mechanistic-descriptive albeit psychological, Brown attempts to link it to the dynamic theories of Freud, Jung and Adler. He strongly favors the analytic schools, but stresses the basic physiology and structural change underlying psychological phenomena. In such an integration the work of McDougall, Pierre Janet and Morton Prince are revitalized and assume renewed importance.

Two complete chapters are devoted to Freud whom Brown calls "the psychological genius of his age" (p. 5). It is conceivable, however, that Freud might have resented Brown's loose use of the word "psychoanalysis" which in this book connotes diversified techniques as word-association and even modified suggestion-persuasion psychotherapy. Furthermore, Brown feels that hypnosis therapy (early abandoned by Freud) can when coupled with post-hypnotic probing into the unconscious be as effective as (and certainly less expensive and time consuming than) formal psychoanalysis. The technique of this method is to hypnotize the patient either lightly or deeply as the case may require and then stimulate him to free association under hypnosis. Insight is forced by hypnotic and post-hypnotic suggestion thus augmenting the therapy produced by the actual psychocatharsis. Brown claims a good deal of success for this method although he is not averse to suiting his technique to the individual. The technique and theory behind it represents psychotherapy but hardly a true "psychoanalysis."

This represents the chief contribution of the book from the standpoint of psychotherapy. The psychological background is adequately handled within the limited available space. A resumé of some theories of emo-

tions, notably the James-Lange theory, is clear and concise. A chapter on psychology and the adolescent, a surprisingly Puritanical outpouring with which there is much to quarrel, is partly compensated for by an excellent chapter on the psychological problems of later life. Sublimation, faith and personal influence (transference) also receive excellent handling. And as is usual in books emanating from England of late there are two chapters dealing with war; one on war neuroses, and the other on the "psychology of peace and war." A philosophical essay on the relation of mind to brain and a short commentary on psychical research (extrasensory perception) complete the book. The appendix contains a description of three cases of war neuroses and a patient's own account of a "psychoanalysis." This particular account is so much a reflection of Brown's personal viewpoint as to suggest that the treatment was more suggestion than it was analysis.

As is apparent from the foregoing the book is packed with provocative material and although there are many who would not agree with much that is said, one has the feeling that the author knows the subject and is sincere in his statements. His therapeutic results can hardly be evaluated but the book appears to be an excellent representation of this particular school of psychiatric thought. As such it is highly recommended.

The Public Health Nurse and Her Patient. By RUTH GILBERT. 396 pp. The Commonwealth Fund, New York, 1940. Price $2.25.

It is difficult to budget time for reading so as to include the many books recently written on health subjects, but there was one published the past year which well deserves attention. It is "The Public Health Nurse and Her Patient," by Ruth Gilbert.

This book is a thorough and detailed description of the way in which a patient is affected by his emotions, by the reactions of the family and by the attitude of the nurse. The attention of the public health nurse is quite properly called to the fact that "we are nursing the patient and not the disease."

Definite techniques for giving practical care to patients have been in use for quite a few years. We could scarcely expect to have such definite techniques outlined for dealing with emotional problems, but Miss Gilbert, nevertheless, has succeeded, in this book, in presenting methods to assist in handling the underlying mental problems which for so many years have handicapped the work of the public health nurse. From her wide knowl-

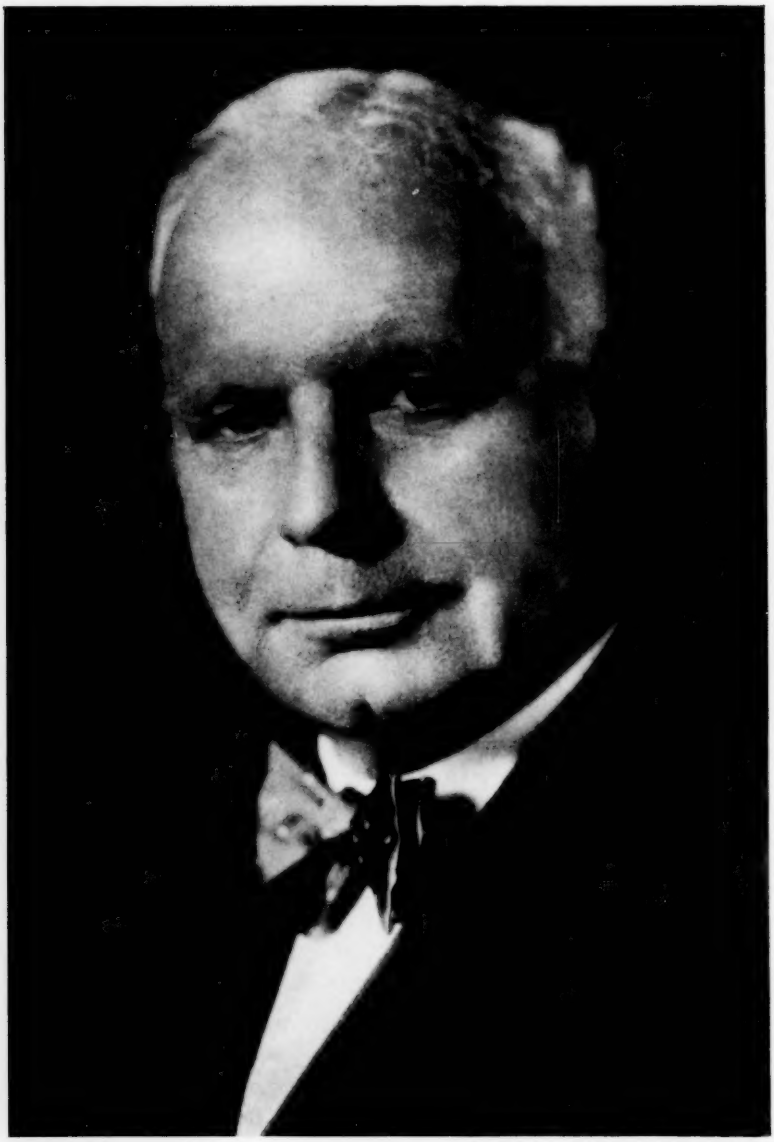
edge of mental hygiene and her extensive experience in family case work, the author has been able to give useful, concrete examples which make her discussion of this subject both practical and interesting.

While this book will no doubt be received more enthusiastically by public health nurses than by other groups, it should prove to be valuable for social workers and even for physicians.

A Review of the Psychoneuroses at Stockbridge. A case study and statistical analysis conducted at Stockbridge, 1936-1939 of histories of patients treated during the years 1910-1934 and an evaluation of the therapeutic results. By GAYLORD P. COON, M. S., M. D., and ALICE F. RAYMOND, A. B. Austen Riggs Foundation, Inc., Stockbridge, Massachusetts, 1940. 299 pages, with appendices. Price not stated.

Those who believe in the efficacy of a statistical approach to the problems of psychotherapy will be gratified by the thoroughness with which that method has been applied in the present study. The authors have taken pains to make clear the bases on which they selected cases and relevant data, and they have not hesitated to indicate sources of possible error. Their work is well done. Their conclusions are tepid. Those who are more interested in a psychiatric study of cases will take pleasure in the excellent abstractions which have been made in the presentation of 92 short summaries of case records. These summaries are arranged in seven groups, following an entirely new plan which makes use of apparent differences in the predominant mechanism at work, rather than of differences in the symptoms.

A large part of the book is devoted to exposition of the chief tenets of treatment, as practised at Stockbridge. A sort of psychiatric eclecticism is implied in the background, from which Dr. Riggs and his associates have synthesized an original concept of reeducation in the treatment of the psychoneuroses; ". . . there is a kinship between its technique and that of the 'persuasive' method of Dubois and the 'psychobiological' approach of Adolf Meyer." (p. 161) and again; "All the maxims of William James pertaining to habit training are utilized, and all the suggestive influence of the physician is mobilized in facilitating the job of rehabilitation." (p. 13). The Freudian type of analysis is not used.



ROBERT WOODMAN, M. D.

ROBERT WOODMAN, M. D.

After 42 years of service in the New York State Department of Mental Hygiene, all spent at the Middletown State Homeopathic Hospital, Dr. Robert Woodman retired as superintendent of that institution on October 31, 1940.

Dr. Woodman was born in Bucks County, Pennsylvania, on January 1, 1875, of parents who were birthright members of the Society of Friends. He attended country schools until he was 15, when he went to the Abington Friends School at Jenkinstown, Penn. Upon graduation from that school, he entered Hahnemann Medical College in Philadelphia, where he completed the prescribed three-year medical course in 1895, at the age of 20, graduating second in his class. Because of his youth, he was not eligible for his degree until the following year, but he accepted an interneship at the Rochester Homeopathic Hospital and remained there for two years. After civil service examination, he was appointed medical interne at Middletown in 1898.

At Middletown, Dr. Woodman was promoted to junior physician a month after his arrival, to assistant physician two years later in 1900, to second assistant physician in 1901 and to first assistant physician in 1902. He became superintendent upon the retirement of Dr. Maurice C. Ashley in 1923.

Dr. Woodman's departmental activities were wide, and he took an active interest in the community life of Middletown, where he will make his home in retirement. Under his superintendency, the physical plant at Middletown had many additions, including Woodman Hall, an infirmary and surgical building of more than 300 beds. He has been chairman of the training school committee and the home and community care committee of the Department of Mental Hygiene, and his methods of placing patients in "family care" have been generally accepted in the department.

At the quarterly conference at the Psychiatric Institute and Hospital on December 21, 1940, an appreciation of Dr. Woodman was given by Dr. John R. Ross, superintendent of the Harlem Valley State Hospital, and a personal friend, as well as a colleague, of Dr. Woodman. Dr. Ross' tribute will be printed in the proceedings of the conference in the January issue of the PSYCHIATRIC QUARTERLY SUPPLEMENT.

Dr. Woodman was married in 1903 to Ethel L. Davis of Middletown, who died in 1925. There are three daughters, Mrs. John N. Eckert of Middletown, Miss Julia Woodman of Orange, N. J., and Mrs. Douglas Smith of Long Branch, N. J.

The QUARTERLY extends its congratulations to Dr. Woodman for his long and successful career and its best wishes for his continued health and prosperity.

WALTER A. SCHMITZ, M. D.

Dr. Walter A. Schmitz, director of clinical psychiatry at Middletown State Homeopathic Hospital since 1928, was appointed superintendent of that hospital on December 1, 1940, succeeding Dr. Robert Woodman, retired.

Dr. Schmitz was born December 27, 1891, in Egg Harbor City, N. J. He was educated in the Philadelphia public schools and at Hahnemann Medical College, from which he was graduated in 1913. He served for a year as house physician at the Yonkers General Hospital in Yonkers, then entered the New York State service. He was promoted to senior assistant physician in 1918 and in that year also was commissioned a first lieutenant in the United States Army medical corps. After army service in the neuropsychiatric division at Camp Upton, he attended the postgraduate course in neuropsychiatry at the Psychiatric Institute in 1920.

Shortly after his army service, Dr. Schmitz selected the equipment and established the Roentgenological laboratory at the Middletown hospital. He has conducted its work ever since and has made further special studies in Roentgenology.

Dr. Schmitz is a member of the American Psychiatric Association and the American Medical Association, and is a diplomate of the American Board of Radiology. In 1917, he was married to Miss Mary M. Norris of Plattsburg.

WALTER A. SCHMITZ, M. D.

NOTES

DR. LANG BECOMES ASSISTANT COMMISSIONER

H. Beckett Lang, M. B., who was appointed superintendent of the Buffalo State Hospital on last July 1, became assistant commissioner of the New York State Department of Mental Hygiene on January 1, 1941. Dr. Lang, a veteran of the first World War and a graduate of the medical faculty of the University of Toronto, has long been in the State hospital service. As he acted as a medical inspector for the Department of Mental Hygiene from February, 1938, until his Buffalo appointment, he has many personal contacts with the institutions throughout the State.

Biographical notes and a photograph of Dr. Lang were published in the July, 1940, issue of the PSYCHIATRIC QUARTERLY at the time of his appointment to the Buffalo superintendency.

—o—

CLARENCE H. PIERCE TAKES POST AS SECRETARY OF MENTAL HYGIENE DEPARTMENT

Clarence H. Pierce, who in the last two years has reorganized the Erie County department of social welfare as its executive director of public assistance, became secretary of the New York State Department of Mental Hygiene on November 4, 1940, succeeding Lewis M. Farrington, who died last August.

Mr. Pierce, who is 33 years old, was graduated from high school in Warren, Pa., in 1925. He attended the University of Michigan from 1925 to 1928 and from 1929 to 1931, receiving his B. A. degree in 1931. The following year, while he was doing graduate work in English literature, he won the Avery Hopwood award of $1,000 for creative writing in the field of drama. Mr. Pierce attended Western State Teachers College at Kalamazoo, Mich., in 1933 and obtained a certificate to teach in secondary schools.

Later, while employed by the Pennsylvania Emergency Relief Bureau from 1934 to 1936, Mr. Pierce was comptroller in Somerset County and in Area 11, which included four counties. He enrolled at the New York School of social work in the summer of 1936 and obtained his certificate the following year. He was appointed assistant professor of public welfare and public administration in the School of Social Work at the University of Buffalo in September, 1937. In May, 1938, Mr. Pierce was appointed to the Erie County position.

Mr. Pierce is married to the former Margaret Anna Henrich of Buffalo. They have a two-year-old son.

DEATH OF DR. PAUL F. SCHILDER

Dr. Paul Ferdinand Schilder, clinical director of the psychiatric division of Bellevue Hospital and an internationally known figure in psychiatric and psychoanalytic circles, died in New York City on December 8, 1940, as a result of injuries suffered when he was struck by an automobile on the evening before. He was 54 years old.

When the accident occurred, Dr. Schilder had just left Doctors Hospital where he had been visiting his wife, Dr. Lauretta Bender, head of the children's ward of Bellevue Hospital and herself a widely known psychiatrist. Ten days previously, she had given birth to the Schilders' third child, a daughter.

Known as a profound scholar and forceful speaker and writer, Paul Schilder had already attained a commanding position in his field when he was brought to America from Vienna in 1929 by Adolf Meyer. In the United States, besides his connection with Bellevue, he became research professor of psychiatry at the College of Medicine of New York University and continued his lecturing and extensive writing. Much of his recent work, particularly with children, had been done in close conjunction with his wife, Dr. Bender.

The Society for Psychotherapy and Psychopathology, of which Dr. Schilder was a founder and the first president, conducted a special memorial meeting for him on December 20. Speakers in tribute to him included Dr. Adolf Meyer, Dr. A. A. Brill, Dr. Karl M. Bowman, Dr. Sam Parker and Dr. Fritz Wittels.

Dr. Schilder wrote voluminously and on subjects covering a wide field, but with much attention devoted to the use and influence of psychoanalysis in psychiatry. He had contributed to the PSYCHIATRIC QUARTERLY. His writings ranged from volumes on *Psychotherapy* and *Brain and Personality* to psychoanalitic studies of Karl Marx and Socialism and of Lewis Carroll and *Alice in Wonderland*.

A sketch of Paul Schilder's life and a bibliography through 1939 appeared in the October number of the Journal of Criminal Psychotherapy, of which Dr. Schilder was an advisory board member. A similar bibliography, which contains more than 200 titles, also appeared in the November issue of Psychiatry.

DR. ROBERT G. COOK DIES

Dr. Robert G. Cook, identified for many years as the principal owner of Brigham Hall Hospital in Canandaigua, died October 25, 1940, in his seventy-sixth year. In the earlier years of his professional life, he had been a member of the staff of the St. Lawrence State Hospital when Dr. P. M. Wise was the superintendent, and later had studied in Great Britain and on the continent.

His father, Dr. George Cook, was a member of the staff of the Utica State Hospital shortly after it was organized and when it was known as the New York State Lunatic Asylum. He resigned in 1855 to establish a private asylum in the western part of the State, becoming associated in this undertaking with Dr. John B. Chapin, who later became the superintendent of the Willard State Hospital and still later of the Pennsylvania Hospital at Philadelphia, where he succeeded Dr. Thomas S. Kirkbride.

"To commemorate the name of Amariah Brigham, the first medical superintendent and organizer of the New York State Lunatic Asylum, the foremost alienist of his day, this hospital bears his name," said Dr. Chapin in an address delivered during the exercises commemorating the semi-centennial of Brigham Hall Hospital.

Dr. Cook enjoyed the esteem and confidence of a wide circle of friends and patrons of Brigham Hall and of his colleagues in the learned professions of western New York.

———o———

THE FIGHT AGAINST POLIOMYELITIS

An event of national importance took place when the first annual medical meeting of The National Foundation for Infantile Paralysis was held in New York City on November 7 and 8, 1940. Basil O'Connor, president of the organization, which was formed early in 1938 to administer the proceeds of the annual celebrations of President Roosevelt's birthday, reported that the foundation had received since its formation, $2,299,000 from the birthday observances and other sources.

Direct expenditures in the fight against this disease of the nervous system have amounted to $1,181,000, 51.37 per cent of the receipts, and have been in the fields of virus research, after-effects research, nutritional research, education, and active combat against epidemics. A notable feature of the foundation's report was the figure of $129,000 cited as the total cost of administration, only 5.61 per cent of the total receipts.

THE QUARTERLY CONFERENCE

The quarterly conference of the New York State Department of Mental Hygiene was held at the New York State Psychiatric Institute and Hospital on December 21, 1940. Dr. Nolan D. C. Lewis, director of the institute, gave a comprehensive review of the research work conducted there. A paper on "Metrazol as an Adjunct in the Treatment of Mental Disorders" was presented by Drs. Clarence O. Cheney, Donald M. Hamilton, and W. Lynwood Heaver; one on "Electric Shock Therapy in Mental Diseases" by Drs. S. Eugene Barrera and Lothar Kalinowsky; and the program also included Dr. John R. Ross' appreciatory address on Dr. Robert Woodman.

———o———

ORTHOPSYCHIATRIC ASSOCIATION MEETING

The American Orthopsychiatric Association, a society for the study and treatment of behavior and its disorders, will conduct its eighteenth annual meeting at the Hotel Pennsylvania, New York City, on February 20, 21 and 22, 1941. Preliminary programs may be obtained from Helen P. Langner, M. D., chairman of the publicity committee, 1790 Broadway, New York City.

———o———

SALMON MEMORIAL LECTURES

Dr. Nolan D. C. Lewis, director of the New York State Psychiatric Institute and Hospital, delivered the eighth series of the Thomas William Salmon Memorial Lectures at the New York Academy of Medicine in November, 1940. Based on the general subject, "The Pathway of Research in Psychiatry," the titles of the three lectures were: "Historical Perspectives of Psychiatric Thought," "Modern Ramifications in Psychiatric Thought and Research" and "Prospects for Future Achievement in Psychiatric Research."